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SUBPHRENIC ABSCESS*

AN ANALYSIS OF 3,372 COLLECTED AND PERSONAL CASES

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Of the late complications of intra-abdominal suppurative processes, subphrenic abscess is one of the most important. Infections of the subphrenic space occur much more frequently than is commonly supposed, but fortunately the majority of such infections will subside spontaneously and may never be diagnosed unless, because of the continuation of the septic manifestations, the possibility of the existence of the lesion is considered.[1] In our experience approximately only 30 per cent. of subphrenic infections actually proceed to suppuration. It is possible that even more cases of subphrenic infection occur which are not diagnosed clinically, but of the cases of subphrenic infection which are diagnosed from the clinical manifestations only 30 per cent. ultimately suppurate; the remaining 70 per cent. subside spontaneously. Neuhof[2] observed among 972 cases of acute appendicitis fifteen cases of subphrenic infection (an incidence of 1.5 per cent.). Similar observations have been made by Lee,[3] Clendening,[4] Ochsner,[5] and McNamee.[6]

The present report includes an analysis of 3,332 cases of subphrenic abscess collected from the world literature and a presentation of fifty additional cases treated in the Charity Hospital and the Touro Infirmary in New Orleans. Whereas a consideration of the racial incidence of subphrenic abscess is not available in the world literature, we have found that of thirty cases admitted to the Charity Hospital, where approximately half of all admissions are colored, fifteen occurred in the colored race. The remaining twenty cases were in Touro Infirmary and other private institutions to which Negroes are not admitted. In our series there were thirty-six (72 per cent.) males and fourteen (28 per cent.) females. The highest incidence in our series was in the fourth decade (32 per cent.) and 70 per cent. occurred between the ages of nine and forty, 6 per cent. being between nine and twelve. (Chart I.)

Subphrenic abscess usually follows a suppurative process within the abdominal cavity. In the series of 3,322 collected cases of subphrenic abscess together with our own series of fifty cases the primary lesion was in the

* Read before the American Surgical Association, May 10, 1933.

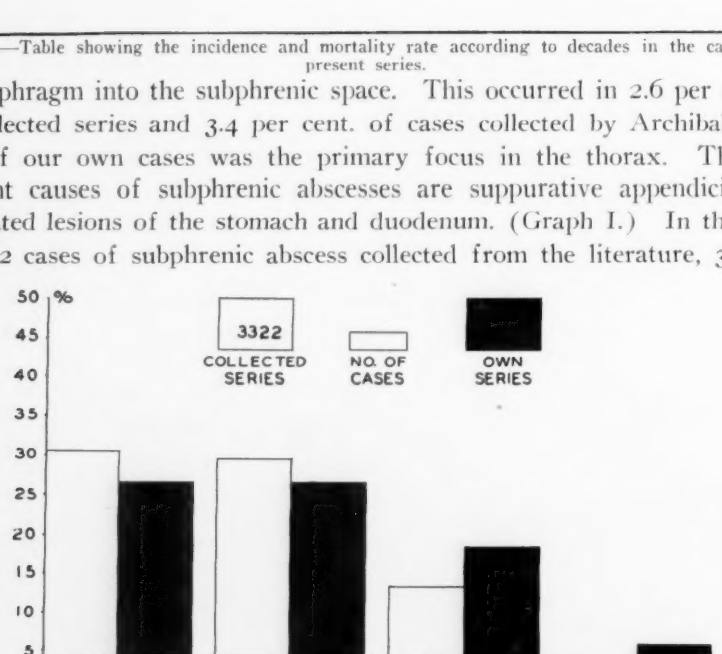
abdomen in 88 per cent. of cases. Exceptionally infection of the subphrenic space may occur as a result of a blood-borne infection, the micro-organisms being transported from some distant focus. This was responsible for but 3.4 per cent. of the collected cases and 6 per cent. of our cases (Chart II). Still less frequently suppurative lesions in the thorax may extend through

Incidence and Mortality Rates According to Decades

	TOTAL NUMBER OF CASES	PERCENTAGE TOTAL GROUP	DIED	PERCENTAGE MORTALITY
9 - 19	10	20%	1	10%
20 - 29	9	18%	3	33%
30 - 39	16	32%	7	43.7%
40 - 49	8	16%	3	37.5%
50 - 59	4	8%	1	25%
60 - 69	3	6%	1	33.3%

CHART I.—Table showing the incidence and mortality rate according to decades in the cases of the present series.

the diaphragm into the subphrenic space. This occurred in 2.6 per cent. of the collected series and 3.4 per cent. of cases collected by Archibald.[7] In none of our own cases was the primary focus in the thorax. The most frequent causes of subphrenic abscesses are suppurative appendicitis and perforated lesions of the stomach and duodenum. (Graph I.) In the series of 3,322 cases of subphrenic abscess collected from the literature, 30.7 per



GRAPH I.—Graphic representation of the comparative frequency of the most frequent lesions in the collected and reported cases of subphrenic abscess.

cent. and 29 per cent., respectively, originated from appendicitis and a perforated lesion of the stomach or duodenum. Therefore, in approximately 60 per cent. of all cases of subphrenic abscess the process originated from the appendix, stomach, or duodenum. In 12 per cent. of the collected series

CHART II.—Etiology in 3,322 collected cases of subphrenic abscess and fifty cases included in the present report.
* This number also includes those cases originating from the gall-bladder, but were not separated in the original report.
** This number includes those cases originating from the pancreas, liver, and intestine which were not separated in the original report.

Etiology

	STOMACH AND DUODENUM	APPENDIX	LIVER AND BILE PASSAGES	SPLEEN	PANCREAS	KIDNEY	TRAUMATIC	FEMALE GENITALIA	METASTATIC OR PRIMARY	INTESTINE	THORAX	MISCELLANEOUS OR UNKNOWN	TUBERCULOSIS	
SCHEURLEN	34 (100%)													
LEJARS		59 (100%)												
MARTINET	55 (36.97%)	8 (5.3%)	25 (16.78%)	6 (4.03%)	3 (2.01%)	4(2.68%)			20 (13.43%)		1 (.67%)	27 (18.12%)		
PIQUAND	267 (32.28%)	191 (21.5%)	136(15.28%)	40 (4.49%)	27 (3.03%)	28(3.15%)	20 (2.26%)	17(1.91%)		20 (2.3%)	32 (3.6%)	69 (7.75%)	23 (2.58%)	
LANG	54 (26.02%)	26 (13.5%)	26(13.5%)	6 (3.13%)				4 (2.08%)	35 (18.23%)		6 (3.13%)	35 (18.23%)		
NORWACK	35 (43.75%)	8 (10%)	11(13.8%)	3 (3.75%)		3(3.75%)	5 (6.25%)	3 (3.75%)			2 (2.5%)	10 (12.5%)		
FINKELSTEIN	77 (30.56%)	45 (17.86%)	26(10.32%)		8 (3.18%)	5(1.98%)	23 (9.13%)	5 (1.98%)		17 (6.8%)	6 (2.4%)	30 (11.9%)	10 (3.97%)	
PERUTZ	70 (33.65%)	55 (26.4%)	22(10.6%)	4 (1.92%)	4 (1.92%)	7(3.4%)	8 (3.9%)	6 (2.9%)	5 (2.4%)	7 (3.4%)	9 (4.3%)	11 (5.29%)		
MADYL	43 (26.7%)	25 (15.5%)	37(22.98%)			11(6.8%)	6 (3.7%)		11 (6.8%)	5 (3.1%)	12 (7.5%)	11 (6.8%)		
GRUNEISEN	10 (16.7%)	27 (45%)	5(8.3%)	5 (8.3%)	1 (1.7%)	4 (6.7%)					6 (10%)	2 (3.3%)		
BARNARD	28 (36.8%)	12 (15.8%)	18(23.7%)	3 (3.95%)	1 (1.3%)	1 (1.3%)		4 (5.3%)	5 (6.6%)	2 (2.6%)		2 (2.6%)		
HUNT	24 (70.6%)	4 (11.8%)	3 (8.8%)	1 (2.94%)		1 (2.94%)	1 (2.94%)							
FLYNN	81* (23.4%)	187 (54.1%)		59** (17.1%)								19 (5.5%)		
DOHERTY AND ROWLANDS	23 (47.9%)	12 (25%)	4(8.3%)						6 (12.5%)	3 (6.3%)				
BEYE	14 (45.2%)	12 (38.7%)	1(3.23%)					2 (6.5%)				2 (6.5%)		
FIFIELD AND LOVE	35 (41.7%)	30 (35.7%)	5(5.95%)	2 (2.4%)		1 (1.2%)	1 (1.2%)					10 (11.9%)		
GATEWOOD	28 (47.5%)	10 (16.95%)	12(20.3%)		1 (1.7%)	3 (5.1%)		4 (6.8%)			1 (1.7%)			
PERRY	8 (17%)	18 (38.3%)	5(10.6%)	1 (2.13%)		1 (2.13%)	3 (6.38%)		6 (12.8%)		2 (4.3%)	3 (6.4%)		
ROSS		31 (100%)												
OCHSNER		19 (100%)												
LOCKWOOD	22 (23.7%)	22 (23.7%)	33(35.5%)						16 (17.1%)					
RUSSEL	2 (9.1%)	6 (27.3%)	6(27.3%)				2 (9.1%)		2 (9.1%)		4 (18.2%)			
WHIPPLE	7 (20%)	5 (14.3%)	10(28.6%)			2 (5.7%)		3 (8.6%)	2 (5.7%)	2 (5.7%)	2 (5.7%)	2 (5.7%)		
ELSBERG		73 (100%)												
MATHER	4 (28.6%)	7 (50%)					1 (7.1%)			2 (14.3%)				
EISENDRATH		34 (100%)												
BROWN	2 (11.1%)	5 (27.8%)	2(11.1%)	1 (5.6%)					1 (5.6%)	1 (5.6%)	1 (5.6%)	5 (27.8%)		
BAUMANN, DEAVER, DOUGLAS CLAIRMONT, AND MEYER, CLENDENING, DEXTER, TUFT, HODGES, McNAMEE, VEDNI, WEBER, SACHS, JORDAN, SCHNITZLER, HOCHENEGG, QUIBAL, ROGER, SCHWARTZ, PANCOAST, MUNRO, and MISCELLANEOUS AUTHORS	21(12.1%)	89 (51.4%)	12(6.9%)			35(20.2%)	3 (1.7%)	2 (1.2%)	3 (1.7%)	2 (1.2%)	2 (1.2%)	4 (2.3%)		
	964(29%)	1020 (30.7%)	399(12%)	131(4%)	45 (1.4%)	106(3.3%)	73 (2.2%)	50 (1.5%)	112 (3.4%)	61 (1.9%)	86 (2.6%)	242 (7%)	33 (1%)	= 3322
OCHSNER AND GRAVES	14 (28%)	13 (26%)	9(16%)				1 (2%)	1 (2%)	3 (6%)			9 (18%)		= 50
GRAND TOTAL	978*	1033	408	131**	45	106	74	51	115	61	86	251	33	= 3372

CHART II.—Etiology in 3,322 collected cases of subphrenic abscess and fifty cases included in the present report.

* This number also includes those cases originating from the gall-bladder, but were not separated in the original report.

** This number includes those cases originating from the pancreas, liver, and intestine which were not separated in the original report.

the infection originated in the liver and bile passages. (Chart II.) Our own cases differ only slightly from the collected ones. Of the fifty cases included in the present report, appendicitis, lesions of the stomach and duodenum, and liver and bile-passage infections were the cause of the subphrenic abscess in 26.1 per cent., 28 per cent., and 18 per cent. of the cases, respectively. (Chart II.)

In a collected series the incidence of subphrenic abscess complicating acute inflammations of the appendix varied from 0.34 per cent. to 6.1 per cent., the average in 11,017 cases of acute appendicitis being 1.1 per cent. (Chart III.) The incidence is undoubtedly higher than these figures indi-

Incidence of Subphrenic Abscess Following Acute Appendicitis as Determined by Cases Collected From the Literature

AUTHOR	CASES ACUTE APPENDICITIS	NUMBER SUBPHRENIC ABSCESSES	PER CENT ACUTE APPENDICITIS
VEGNI	368	5	1.3
ROSS	3391	31	0.7
ELSBERG	98	2	2.
STILLMAN	545	9	1.65
BANCROFT	584	2	0.34
CUTLER	974	6	0.61
BEEKMAN	145	1	0.7
CLAIRMONT AND MEYER	1179	7	0.5
SUERMONDT	630	39	6.1
DEAVER	1700	12	0 .7
WEBER	300	9	3.
DEWES	103	2	2.
STEICHELE	1000	5	0.5
TOTAL	11017	130	1.1%

CHART III.—The incidence of subphrenic abscess following acute appendicitis as determined by cases collected from the literature.

cate, because in many cases the subdiaphragmatic complication is not even suspected, much less diagnosed. Obviously in perforated appendicitis the incidence is higher. Brown[8] observed two cases of subphrenic abscess among the 113 cases of perforated peptic ulcer operated upon at the Presbyterian Hospital in Philadelphia, an incidence of 1.7 per cent.

The micro-organisms responsible for subphrenic infections vary according to the original infection. The micro-organisms most frequently obtained from subphrenic abscesses are the colon bacillus, streptococcus, and staphylococcus. *B. coli* were present in 25 per cent., 30.2 per cent., and 16 per cent., respectively, of Barnard's,[9] Whipple's,[10] and Beye's[11] cases. The same authors found streptococci in 8.5 per cent., 20.9 per cent., and 54 per cent. of their cases, respectively. In our own series in which positive cultures were obtained, *B. coli* were present in 40 per cent., streptococci in 40 per cent., and staphylococci in 20 per cent.

Micro-organisms may gain entrance to the subphrenic spaces in a number of different ways. The infection may extend intraperitoneally, extraperitoneally, or through vascular channels from a neighboring or from a distant focus as follows:

(1) Obviously the simplest mode of infection is the local invasion of the subphrenic spaces by micro-organisms from lesions in the immediate vicinity.

(2) Peritoneal exudate from distant portions of the peritoneal cavity (right iliac fossa and pelvis) may drain into the subphrenic area. With the patient in the supine position the two lowest portions of the peritoneal cavity are the cul-de-sac of Douglas and the posterior portion of the subphrenic space, the general abdominal cavity being divided into these two areas by the ventral curvature of the lumbar spine. As suggested by Eisendrath[12] and Nather[13] the infection may extend from the right iliac fossa through the gutter between the ascending colon and the lateral parietal peritoneum to the region of the right kidney and thus gain entrance to the subphrenic space.

(3) Retroperitoneal phlegmon: As a result of infection of the retroperitoneal cellular tissue a phlegmon extending upward to the extraperitoneal subphrenic area may occur.

(4) Retroperitoneal lymphangitis: Infections may extend through the retroperitoneal lymphatics to the subphrenic spaces. This mode of infection has been especially emphasized by Munro.[14]

(5) Lymphangitis of lymph vessels accompanying the deep epigastric artery. (Barnard.[15])

(6) Rupture of a liver abscess (usually following suppurative portal thrombophlebitis) into the subphrenic space.

Ullman and Levy[16] believe that it is possible to determine the route of infection from the location of the subphrenic abscess. Those infections which occur as a direct extension are located intraperitoneally, whereas those which follow infections of the cellular tissues are retroperitoneal, and those which extend by the lymphatic system are either intraperitoneal or retroperitoneal.

To Martinet,[17] in France, and Barnard,[9] in England, belongs credit for accurately describing the subphrenic spaces. Because so frequently infrahepatic and suprahepatic abscesses occur concomitantly, it is desirable to consider the subphrenic area as that space bounded above by the diaphragm and below by the transverse colon and transverse mesocolon. (Fig. 1.) The anatomy of this area which has been described in detail in previous publications [5, 18, 19, 20] is briefly as follows: The area between the diaphragm above and the transverse colon and transverse mesocolon below is roughly divided by the liver into suprahepatic and infrahepatic spaces which in turn are subdivided into right and left spaces. The suprahepatic area is divided into right and left portions by the coronary ligament, which is the reflection of the peritoneum from the undersurface of the diaphragm on to the superior surface of the liver and the falciform or suspensory ligament, the lower free edge of which extends to the umbilicus as the round ligament. (Fig. 2.) On the superior surface of the liver there are three intraperitoneal spaces and one extraperitoneal space, the extraperitoneal space being located, as the name implies,

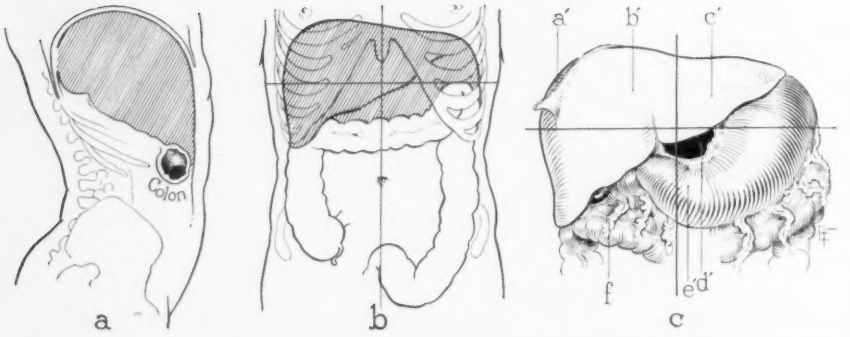


FIG. 1.—(a) The shaded area represents the subphrenic space bounded above by the diaphragm and below by the transverse colon and transverse mesocolon. (b) Subphrenic space, shown in (a), is subdivided by the liver into suprahepatic and infrahepatic portions. (c) Diagrammatic drawing showing the location of the various subphrenic spaces. (a') Right posterior superior space located above the liver and behind the right lateral ligament which is the right prolongation of the coronary ligament. (b') The right anterior superior space located above the liver and anterior to the right lateral ligament. (c') The left superior space located between the liver and the left hemidiaphragm. On the left, because the left lateral ligament courses posteriorly, there is only one space in contrast to the two spaces on the right. (d') The left posterior inferior space or lesser sac located below the liver and behind the gastrohepatic omentum and stomach. (e') The left anterior inferior space located below the liver on the left side and anterior to the stomach. (f') The right inferior space located to the right of the round ligament and the ligament of the ductus venosus.

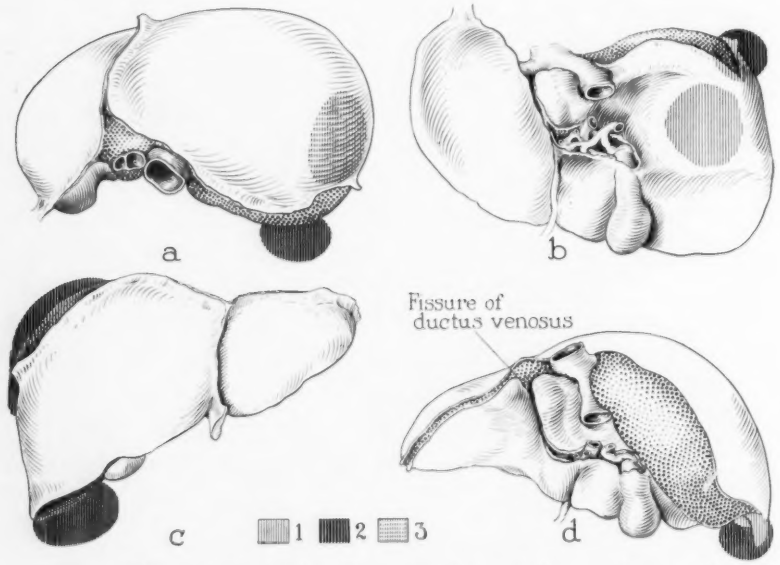


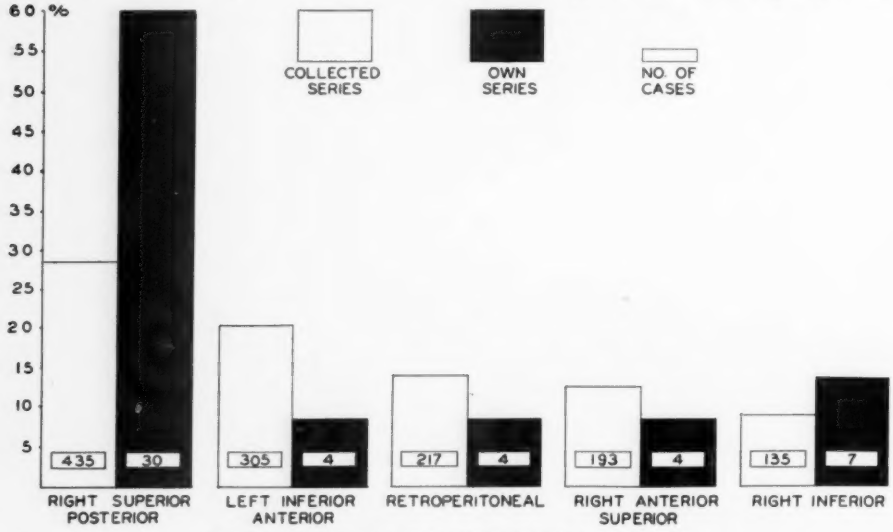
FIG. 2.—Diagrammatic drawing illustrating the most frequent locations for subphrenic abscesses, *viz.*, in the right posterior superior and right inferior spaces. (Key to figure.—(1) Visible abscess areas. (2) Abscesses invading peritoneal cavity. (3) Invisible abscess areas.) (a) Right inferior abscess is shown through the liver. Right posterior superior abscess is shown as abscess invading the peritoneal cavity above the liver and behind the right lateral ligament. (b) Liver viewed from below, showing right inferior space abscess and abscess from right posterior superior space invading the peritoneal cavity. (c) Liver viewed from in front, showing right inferior space abscess below liver and the right posterior superior abscess above the liver and behind the right lateral ligament. (d) Liver viewed from behind, showing typical location of right posterior superior space with abscess above the liver and behind the right lateral ligament.

Location of Abscesses

	RT. POSTERIOR SUPERIOR	RIGHT INFERIOR	RT. ANTERIOR SUPERIOR	RETROPERITONEAL	LEFT ANTERIOR INFERIOR	LEFT POSTERIOR INFERIOR	LEFT SUPERIOR	COMBINED	NOT DETERMINED
MARTINET	54 (41.53%)	4 (3.07%)		18 (13.84%)	38 (29.23%)	4 (3.07%)			12 (9.23%)
PIQUAND	272 (27.55%)	69 (6.98%)	141 (14.28%)	151 (15.30%)	211 (21.37%)	31 (3.14%)	47 (4.76%)	46 (4.66%)	19 (1.91%)
DEXTER	4 (66.67%)		1 (16.66%)	1 (16.66%)					
GATEWOOD									9 Left (45.65%) and 32 Right(54.45%)
FIFIELD AND LOVE	29 (25.89%)	29 (25.89%)	13 (11.60%)	20 (17.85%)	14(12.5%)	7 (6.25%)			
SCHWARTZ	5 (55.55%)	1 (11.11%)	1 (11.11%)		1 (11.11%)		1 (11.11%)		
BARNARD	10 (10.75%)		27 (29.03%)	23 (24.73%)	30 (32.36%)	3 (3.23%)			
PANCOAST	8 (50%)		1 (6.25%)	4 (25%)	2 (12.50%)		1 (6.25%)		
DOHERTY AND ROWLANDS	3 (42.86%)	1 (14.28%)	1 (14.28%)		1 (14.28%)		1 (14.28%)		
DOUGLAS	5 (62.50%)	3 (37.50%)							
McNAMEE	3 (33.33%)	3 (33.33%)			1 (11.11%)		2 (22.22%)		
EISENDRATH	3 (33.33%)	4 (44.44%)			1 (11.11%)		1 (11.11%)		
OCHSNER	19 (82.61%)	4 (17.39%)							
MUNRO	3 (37.5%)	2 (25%)			2 (25%)	1 (12.5%)			
ROSS	13 (40.63%)	14 (43.75%)			1 (3.12%)		2 (6.25%)	2 (6.25%)	
NATHER	4 (19.05%)	1 (4.76%)	8 (38.09%)		3 (14.29%)	1 (4.76%)		4 (19.05%)	
TOTAL	435 (28.86%)	135 (8.9%)	193 (12.7%)	217 (14.4%)	305 (20.2%)	47 (3%)	55 (3.6%)	52 (3.4%)	72 (4.7%)
OCHSNER AND GRAVES	30 (60%)	7 (13.5%)	4 (8%)	4 (8%)	4 (8%)		1 (2%)	4 (8%)	
GRAND TOTAL	464	142	197	221	309	47	56	56	72

CHART IV.—Localization of subphrenic abscesses in 1,517 collected cases and fifty cases included in the present report.

extraperitoneally within the confines of the coronary ligament. On the right side the right lateral ligament, which is the right prolongation of the coronary ligament, divides the area into two spaces, a large anterior one and a relatively small posterior one, being, respectively, the right anterior superior and the right posterior superior spaces. The left lateral ligament coursing along the posterior border of the left lobe of the liver separates the superior surface from the inferior surface of the liver. In the left suprahepatic area there is only one space, the left superior. In the infrahepatic area there are three intraperitoneal spaces which are divided into right and left portions by the round ligament and the ligament of the ductus venosus. To the right of these structures is large space known as the right inferior space. To the left are two spaces separated from each other by the stomach and the gastrohepatic omentum, the anterior one being the left anterior inferior space and the posterior one being the left posterior inferior space or lesser peritoneal sac. The space most frequently involved in subphrenic infections is the right posterior superior space, probably because the most frequent cause of subphrenic infection is suppurative appendicitis. The right posterior



GRAPH II.—Graphic representation of the most frequent sites of localization of subphrenic abscesses in the collected and reported cases.

superior space is the earliest involved because the inflammatory exudate travels upward from the right iliac fossa along the paracolic groove. In a series of 1,517 cases collected from the literature in which localization of the abscesses was stated, the right posterior superior space was involved in 28.8 per cent. In our own series of fifty cases this space was involved in thirty (60 per cent.). (Chart IV.) In addition to abscesses in the above-described subphrenic spaces, retroperitoneal abscesses may dissect upward between the diaphragm and diaphragmatic peritoneum and thus become retroperitoneal subphrenic abscesses. In the collected series, abscesses in the retroperitoneal spaces (14.4 per cent.) followed the right posterior superior space infections in frequency, whereas in our series the right inferior space was involved in 13.5 per cent. of the cases. (Graph II.) In four (8 per cent.) of our fifty cases, abscesses were found in both the right posterior superior and the right inferior spaces. Similar association of infection of these two spaces has been observed by Nather and Ochsner,[18] Strauss,[21] and Milloy.[22] Intraperitoneal subphrenic abscesses occur more frequently than the extraperitoneal. In Elsberg's[23] series of seventy-three cases the lesion was extraperitoneal in 27 per cent., intraperitoneal in 48 per cent., and doubtful in 25 per cent. of the cases.

The clinical picture in subphrenic abscess in general is one of continued infection. If a patient who has had an antecedent suppurative intraperitoneal process fails to improve as he normally should and in whom no other focus can be demonstrated to account for the septic manifestations, one must consider subphrenic infection as a possible cause until proved otherwise. Only by keeping the condition in mind and looking for other diagnostic evidences of this suppurative process which is located in such an inaccessible portion of the peritoneal cavity can an early diagnosis be made. Undoubtedly the inaccessibility of the subphrenic region is responsible for the delay in diagnosis in many cases. The fact that subphrenic infections may exist for long periods of time and produce few or no characteristic manifestations and not be recognized is exemplified by the following: Russel[24] reports three cases of subphrenic abscess in which diagnoses were made seven months after perforation of a peptic ulcer, one year after pneumonia, and seven years after an empyæma. Lockwood[25] reports a case in which the diagnosis was not made until twenty months after the original operation, and Grove[26] reports two cases in which the diagnoses were made one and a half years and three and a quarter years, respectively, after the original intraperitoneal suppuration. Barnard[9] and Whipple[10] suggest that the onset of symptoms in cases of subphrenic abscess is usually one of three types as follows:

(1) Sudden abrupt onset with symptoms simulating an acute intra-abdominal suppuration. These are usually cases in which the causative agent, such as perforated peptic ulcer, perforated appendicitis, *etc.*, results in massive contamination of the peritoneal cavity. Thirty-four and three-tenths per cent. of Whipple's[10] and 62.6 per cent. of Bernard's 9 cases were of this type.

(2) Insidious onset usually following an obscure intra-abdominal lesion. These cases are frequently not suspected and, therefore, not diagnosed. In 40.6 per cent. of Whipple's[10] cases the onset was of this type.

(3) Following laparotomy for an intraperitoneal suppurative process, at which time the subphrenic space was uninvolved, the septic manifestations continue and the patient does not improve. Forty-seven and three-tenths per cent. of Barnard's[9] cases were of the last two types and 25 per cent. of Whipple's[10] cases were of the third type.

In our experience most cases have followed a known intraperitoneal suppuration. Of the fifty cases herein reported, the onset was sudden in eight (16 per cent.), insidious in seven (14 per cent.), whereas in thirty-five (70 per cent.) cases systemic manifestations continued following drainage of the original suppurative process. In this last group, even though the original suppurative lesion had been treated correctly, systemic manifestations of infection persisted. Pyrexia and leucocytosis continued, and the patients exhibited other signs of continued infection. In addition to these systemic signs there may or may not be localizing manifestations. Occasionally there will be a sense of pressure in the upper abdomen or loin, and difficulty in breathing, especially on deep inspiration, may be complained of. In those

individuals with an infection of the right posterior superior space, the pain, when present, is referred to the lumbar region, whereas in those cases with right anterior superior space or right inferior space infections the pain is referred to the right costal margin. Limitation of respiratory movements on the affected side occurs relatively early. The diaphragm is elevated and its excursion diminished. Of greatest diagnostic importance is persistent, localized tenderness over the involved portion. If the abscess is in the right posterior superior space, there is persistent, localized tenderness over the right twelfth rib, which may be the only sign. The tenderness is localized along the costal margin, on their respective sides, in right anterior superior space, right inferior space, left superior space, and left anterior inferior space abscesses. If the tenderness persists together with continued systemic manifestations of unabating infection, one is justified in diagnosing a subphrenic infection of the particular space involved; and if, under conservative therapy, the symptoms and signs do not subside, an exploration of the space is justified. In right anterior superior space, right posterior superior space, and left superior space abscesses the liver is displaced downward and can be felt extending for varying distances below the costal margin. The area of liver dulness is definitely increased.

The röntgenological findings are of diagnostic importance. Elevation and immobility of the diaphragm have been especially emphasized by Lewald,[27] Pancoast,[28] Granger,[29] O'Brien,[30] Schwartz,[31] and McNamee.[6] In our series röntgenograms were obtained in forty. In thirty-three (80 per cent.) there was an elevation of the diaphragm. Pancoast[28] and Granger[29] stress the fact that even though röntgenological findings are of utmost importance as regards diagnosis of subphrenic infection, it is important and essential to correlate these findings with the clinical data in order that the correct diagnosis might be made. Röntgenograms should be made with the patient in the upright position and, as suggested by Granger,[29] preferably at a distance of six feet. Two views, an anterior posterior and a lateral one, should always be obtained. Granger[29] differentiates between subphrenic abscesses resulting from perforation of a liver abscess and those in which infection occurs as a result of a suppurative process within the peritoneal cavity as follows: Röntgenograms of subphrenic abscesses following rupture of a liver abscess show obliteration of the cardiophrenic angle in the postero-anterior view and obliteration of the anterior costophrenic angle in lateral view, whereas röntgenograms of subphrenic abscesses following intraperitoneal infections show obliteration of the costophrenic (instead of the cardiophrenic) angle in the anteroposterior view, and an obliteration of the posterior (instead of the anterior) costophrenic angle in the lateral view. This is possibly due to the fact that the most frequently involved space following an intra-abdominal suppurative process is the right posterior superior one. As the presence of gas in subphrenic abscesses represents a late finding, its absence does not in any way eliminate a diagnosis of subdiaphragmatic suppuration. Unfortunately, in text-books and many other publications the importance of röntgenological demonstration of gas with the production of a typical fluid

level has been emphasized. When gas is present, it is, of course, of diagnostic importance, but as in the majority of cases gas is not present and in practically all represents a late finding, its absence is of little significance. According to Elsberg[23] and Berman,[32] only 15 per cent. of subphrenic abscesses contain gas. Gas is more apt to be demonstrated in those cases in which the antecedent lesion was a perforated peptic ulcer or perforation of some other portion of the gastro-intestinal tract. Its presence may be accounted for in this way or may result from the production of gas by gas-producing micro-organisms. In those cases in which gas is present in the abscess Whipple,[10] Sommer,[33] and Meller[34] advocate taking röntgenograms in various positions in order to show the extent of and more accurately localize the abscess. By so doing Sauer[35] was able to diagnose three separate abscess cavities in a patient of his.

The attempted aspiration of pus from a subphrenic abscess is to be condemned, because of the danger of contaminating uninvolved portions of the pleural or peritoneal cavities. Barnard[9] reported a case in which, following the transpleural aspiration of a subphrenic abscess, the patient collapsed and died three hours later. At autopsy one and a half pints of pus were found to have leaked into the pleural cavity and this undoubtedly was the cause of the patient's death. The following are opposed to aspiration of these abscesses: Whipple,[10] Ochsner, [5, 19, 20] Graf,[36] Grove,[37] Schwartz,[31] and Doherty and Rowlands.[38] Even those surgeons who advocate aspiration of the subphrenic area in order to attempt to determine the presence of pus emphasize that such a procedure should be done directly before operation so that if pus is found, immediate incision and drainage might be instituted. We are convinced from our own personal experience and also based upon sound surgical principles that if aspiration of the subphrenic space is to be attempted it should be made in such a way that uninvolved portions of the pleura and peritoneum are not traversed with the aspirating needle. This can be accomplished, especially in infections of the right posterior superior space, by introducing the needle below the twelfth rib and extending upward and forward. It is important when this is done to have the syringe attached to the needle and to maintain aspiration during the introduction of the needle. Only in this way can one be relatively certain of not traversing an encapsulated suppurative process and penetrating beyond it. Occasionally such will occur even though one aspirates during the introduction of the needle due to the plugging of the needle with thick exudate. We, however, believe it is distinctly safer and, therefore, much better to explore the subphrenic space under local analgesia as only in this way can one be certain that contamination of one of the serous cavities does not occur.

Intrathoracic inflammatory processes are the most frequent complications of subphrenic abscess and when present usually mask the clinical picture. The incidence of intrathoracic complications varies according to different authors and undoubtedly varies with the length of time which elapses between the development of the subphrenic infection and the institution of therapy. The majority of clinicians are of the opinion that subphrenic

Complications

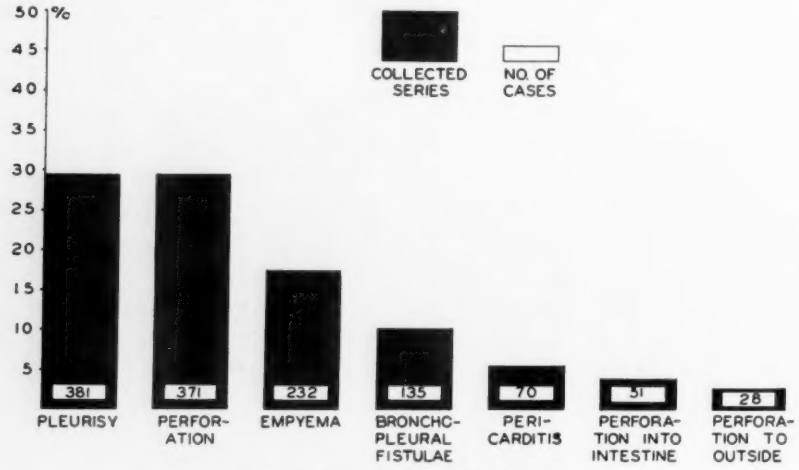
	PERFORATIONS	PLEURISY	EMPYEMA	BRONCHO-PLEURAL FISTULA	PERICARDITIS	MEDIASTINAL ABSCESS	GENERAL PERITONITIS	PERFORATION INTO INTESTINE	PERFORATION TO OUTSIDE	PNEUMONIA	PNEUMOTHORAX	LUNG ABSCESS
FENWICK	12 (85.72%)								2 (14.28%)			
BEYE		2 (9.09%)	10 (45.45%)	6 (27.47%)						(18.28%)		
LANG	67 (80.72%)				16 (19.28%)							
LOCKWOOD	4 (22.22%)		11 (61.11%)									3(16.7%)
MARTINET	33 (100%)											
PIQUAND	175 (25.56%)	199 (28.96%)	81 (11.84%)	112(16.37%)	41 (5.99%)	2 (2.90%)	16 (2.32%)	34 (4.97%)	24 (3.51%)			
FINKELSTEIN	74 (41.57%)	45 (25.28%)	46 (25.84%)		13(7.30%)							
DEXTER		3 (60%)	1 (20%)	1 (20%)								
GATEWOOD	1 (5.85%)	2 (11.70%)	11 (64.10%)				1 (5.85%)			1 (5.85%)		1(5.85%)
FIFIELD AND LOVE	5 (33.33%)		4 (26.66%)					4 (26.66%)				2(13.3%)
WHIPPLE		4 (19.04%)	10 (47.61%)							7 (33.32%)		
PENUTZ		60 (100%)										
GRUNEISEN		40(97.55%)					1 (2.43%)					
SCHWARTZ			4 (80%)	1 (20%)								
BARNARD		6 (20.69%)	5 (17.24%)	4 (13.79%)			1 (3.45%)	11 (37.93%)	2 (6.89%)			
PANCOAST			1 (100%)									
DOHERTY AND ROWLANDS			3 (75%)								1 (25%)	
ELSBERG		8 (21.05%)	22 (57.89%)	6 (15.79%)						1 (2.63%)		1(2.63%)
DOUGLAS			2 (66.66%)	1 (33.33%)								
McNAMEE		1 (16.66%)		1 (16.66%)				1 (16.66%)		1 (16.66%)		2(33.3%)
EISENDRATH			4 (100%)									
MUNRO			2 (66.66%)					1 (33.33%)				
NATHER		3 (50%)	1 (16.66%)									2(33.3%)
ROSS		3 (25%)	6 (50%)	2 (16.66%)						1 (8.33%)		
TOTAL (COLLECTED)	371(29%)	376 (29%)	224(17.5%)	134 (10%)	70(5.3%)	2 (0.2%)	19 (1.4%)	51 (3.8%)	28 (2.3%)	15 (0.9%	1(0.1%)	11 = 1302 (0.8%)
OCHSNER AND GRAVES		5 (25%)	8 (40%)	1 (5%)						4 (20%)	2 (10%)	= 20
GRAND TOTAL	371	381	232	136	70	2	19	51	28	19	3	11 = 1322

CHART V.—Complications and incidence in collected and reported cases of subphrenic abscess.

infections are frequently complicated by extension to the thorax. According to Clute[39] and Beye,[11] the thoracic involvement usually consists of an irritative pleuritis which results in a serious effusion. We cannot agree with Beye[11] that one of the earliest findings in subphrenic abscess is pleural effusion or with Clute[39] that "it is almost always true that a simple serous pleurisy will be present in the chest when there is pus just beneath the diaphragm." Conversely, we believe that extension of the infection to the thorax usually is the result of a late diagnosis of the subphrenic infection which has existed long enough for the micro-organisms and toxins to have passed through the diaphragmatic lymphatics into the pleural cavity. We are convinced that if an early diagnosis of subphrenic abscess is made and the correct therapy instituted the incidence of intrathoracic complications can be materially reduced. We agree with Dexter[40] that "obviously it is highly desirable to drain the abscess before the structures above the diaphragm are involved." That subphrenic abscesses can be diagnosed before intrathoracic complications occur is shown by the fact that in the personally treated cases none of these complications occurred. Of thirty-one cases of subphrenic infection reported by Beye,[11] thoracic complications occurred in twenty-three (71.4 per cent.). Beye's[11] statistics suggest, however, that the diagnoses in his cases were made relatively late because of the twenty-three cases with thoracic complications, fifteen (62.5 per cent.) had a gross perforation of the diaphragm. In Lockwood's[25] eighty-two cases of subphrenic abscess operated upon at The Mayo Clinic intrathoracic complications occurred in 20.7 per cent. In addition to simple serous effusion which may be of little or no consequence as regards the subsequent clinical course, but which may be of great importance as regards the difficulty in diagnosing the condition, there may be other intrathoracic complications such as bronchopleural fistula, lung abscess, and pneumonitis. (Chart V. Graph III.) The manner in which pneumonitis and infections of the pulmonary parenchyma might occur without involvement of the pleura is suggested by Menville's[41] and Schlanger's[42] observations. Menville was able to demonstrate in both animals and humans that following the intraperitoneal injection of thorium dioxide, extension to the bronchial and retrosternal lymph-nodes occurred. Schlanger[42] found that röntgenograms taken following the filling of a subphrenic abscess cavity with lipiodol showed lymphatic channels extending retroperitoneally and retropleurally to the hilum of the lung.

The prognosis in subphrenic abscess is dependent upon a number of factors, chief among which are the length of time elapsed from the beginning of the infection to the institution of therapy, the presence of complications, and the treatment instituted. Everything else being equal, the earlier the recognition of a subphrenic abscess and the earlier correct drainage is instituted, the better is the prognosis. As a result of delayed diagnosis the patient may die of sepsis. The importance of thoracic complications as regards prognosis is exemplified by the following statistics. Of the thirty-

one deaths in Lockwood's[25] series, thirteen (41.9 per cent.) had intrathoracic complications. The mortality rate in Beye's[11] cases with thoracic complications was 43.5 per cent., whereas the mortality rate in the cases with no complicating thoracic lesion was 25 per cent. Of the seven cases which developed empyæma in Gatewood's[43] series, five died. In our own series the mortality rate in those cases with thoracic complications was 52 per cent., whereas in those with no thoracic complications the mortality rate was 18 per cent. The mortality rates in the Charity Hospital series and in the private cases in our series were approximately equal, 32.2 per cent. in the former and 31.5 per cent. in the latter. The race and sex were of no prognostic importance, the mortality rates being equal in the white and colored races and in both sexes. The mortality rate was highest in the fourth decade (43.7 per cent.) and lowest in the second decade (10 per cent.) (Graph IV. Chart I.)



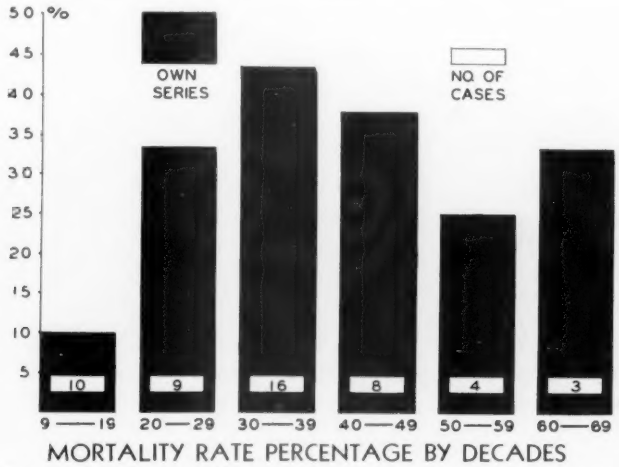
GRAPH III.—Complications and incidence in order of their frequency in the collected cases of subphrenic abscess.

Whereas non-operative conservative treatment is indicated in cases of subphrenic infections which have not progressed to suppuration, the treatment of a subphrenic suppurative process is incision and drainage. Of 1,072 reported cases of subphrenic abscess in which non-operative treatment was used, the mortality rate was 91.1 per cent. as contrasted with the mortality rate of 33.6 per cent. in 1,693 cases in which drainage was instituted. All the cases in our series were operated upon with a general mortality rate of 32 per cent. (Chart VI. Graph V.)

The prognosis in subphrenic abscess depends not only upon the institution of drainage but also upon the type of drainage employed. If in draining the abscess a contamination of an uninvolved serous cavity occurs, obviously the prognosis is much worse both as regards life and subsequent morbidity than it would be if such a contamination did not occur. In the collected series of 189 cases of subphrenic abscess drained without contamination of the pleural or the peritoneal cavities, the mortality rate was 21 per

cent., whereas of 305 cases drained transpleurally 39 per cent. died, and of 307 drained transperitoneally 35.5 per cent. died. In our own series the mortality rates following extraperitoneal, transpleural, and transperitoneal drainages were 13.6 per cent., 50 per cent., and 41.6 per cent., respectively. (Chart VI. Graph V.)

Treatment.—The treatment of subphrenic abscess is entirely surgical and as in most pyogenic suppurative processes the treatment consists of incision and drainage. It must, however, be emphasized that the majority of subphrenic infections do not progress to suppuration and that in those cases in which a subphrenic infection is diagnosed before suppuration has occurred, conservative treatment should be instituted and continued until either suppuration has occurred or spontaneous resolution has taken place. This can usually be easily determined by the clinical course. The necessity for surgi-



GRAPH IV.—The mortality rate percentages by decades in the cases of subphrenic abscesses reported in the present communication.

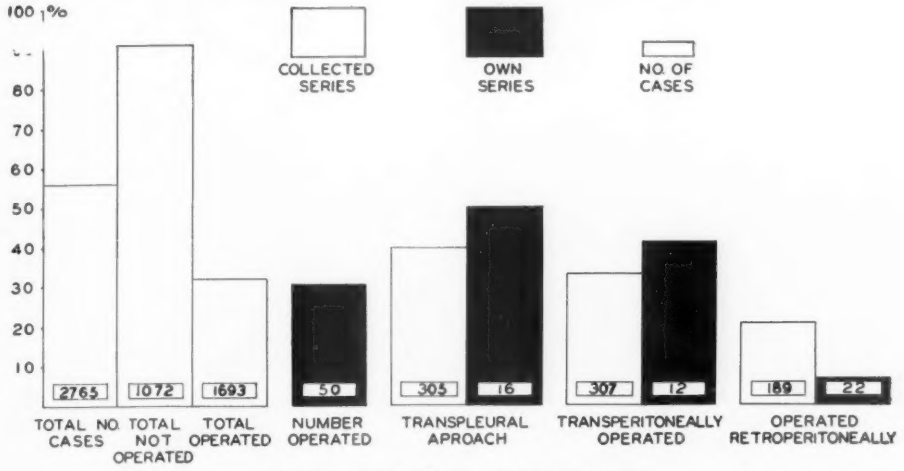
cal drainage in cases of subphrenic suppuration is emphasized by the mortality statistics quoted above. As already mentioned, the mere drainage of a subphrenic suppurative process, however, is not sufficient in order to treat the patient properly. Here, as elsewhere, it is of utmost importance to avoid unnecessary contamination of uninvolved areas. Of especial importance is the avoidance of contamination of an uninvolved serous cavity as it is a well-known fact that the flooding of a virgin serous cavity with toxic material, because of the marked absorbability of serous membranes, is apt to produce systemic symptoms and even death of the individual. Everything else being equal, it is imperative, therefore, to drain a subphrenic suppurative process in such a way that uninvolved portions of either the pleural or peritoneal cavity are not contaminated. This fact should be emphasized because even at the present time this surgical principle has been more or less disregarded in the treatment of these suppurative processes. Barnard,[9] in 1908, considered that of thirty-six deaths from subphrenic abscess reported by him twenty-four were avoidable. In regard to this he states: "In

Mortality Rate Percentages

	TRANSPERITONEAL			TRANSPLEURAL			RETROPERITONEAL			TOTAL GROUP			OPERATED			NON-OPERATED		
	Total	Died	Morta...y	Total	Died	Mortality	Total	Died	Mortalit,	Total	Died	Mortality	Total	Died	Mortality	Total	Died	Mortality
FIFIELD AND LOVE	21	5	23.8%	32	14	43.7%	6	1	16.7%	76	39	50%	59	20	32%	19	19	100%
FLYNN							38	7	18.4%	313	94	30%	313	94	30%			
DOUGLAS	2	0	0	3	1	33%	5	1	20%	11	2	18%	10	2	20%	1	0	0
GATEWOOD										41	14	34%	38	11	28%	3	3	100%
DOHERTY AND ROWLANDS				4	3	75%	2	0	0	6	3	50%	6	3	50%			
DEXTER										6	2	33%	6	2	33%			
BARNARD	39	15	38%	16	6	37.5%	18	6	33.3%	76	36	47%	64	24	37.5%	12	12	100%
BERMAN	1	0	0				1	0	0	2	0	0	2	0	0			
TUFT	2	0	0	1	1	100%	1	0	0	6	3	50%	4	1	25%	2	2	100%
LOCKWOOD										113	58	51.3%	81	27	33.3%	32	31	96.8%
MICHEL AND GROSS										44	34	77.2%	19	9	47.3%	25	25	100%
SCHWARTZ				3	2	66.6%	4	1	25%	8	4	50%	7	3	42.8%	1	1	100%
PERUTZ										208	82	40%	155	38	25%	53	44	83%
MADYL										178	133	74%	74	35	48%	104	98	94%
GRUNEISEN										60	20	33%	60	20	33%			
MUNRO	3	1	33%	1	1	100%				8	6	75%	4	2	50%	4	4	100%
ROSS	12	5	41%	6	5	83%	2	0	0	33	23	70%	20	10	50%	13	13	100%
EISENDRATH				3	3	100%	1	0	0	34	11	32.6%	33	10	30.3%	1	1	100%
McNAMEE	3	0	0	5	2	40%	1	0	0	15	5	33%	13	4	30%	2	1	50%
PANCOAST	1	1	100%				10	3	34.5%	11	2	18%	11	2	18%			
ELSBERG				22	8	36.3%	21	3	14.2%	73	29	40%	51	11	22%	22	18	82%
OCHSNER							19	1	5.25%	19	1	5.25%	19	1	5.25%			
BROWN										18	8	44.4%	18	8	44.4%			
PIQUAND	219	81	37%	206	70	34%	52	13	25%	890	532	60%	477	164	30%	413	368	87%
LOWENSTEIN										104	98	94.2%				104	98	94.2%
LANG										80	71	88.75%				80	71	88.75%
FINKELSTEIN										252	171	70%	134	59	44%	118	112	94.06%
MARTINET										63	56	88.8%				63	56	88.8%
NATHER	4	2	50%	3	3	100%	8	4	50%	15	9	60%	15	9	60%			
TOTAL (COLLECTED)	307	110	35.5%	305	119	39%	189	40	21.17%	2765	1546	56%	1693	569	33.6%	1072	971	91.14%
OCHSNER AND GRAVES	12	5	41.6%	16	8	50%	22	3	13.6%	50	16	32%	50	16	32%			

CHART VI.—Mortality rate in 2,796 collected cases and fifty cases included in the present report according to the type of therapy, whether operative, non-operative, and if operative according to the type of operation used.

short, half the avoidable deaths may be attributable to the fact that the condition was not recognized at all, the other half to the absence of exact and detailed knowledge of the localization and character of subphrenic abscesses on the part of the surgeons when they were planning or executing their operations." The high mortality rate following operations of subphrenic abscess is still undoubtedly due to the conditions which were suggested by Barnard,[9] in 1908. Because suprahepatic abscesses, even though they are located within the abdomen, are covered by the thoracic cage and because the most direct route to these abscesses is through the thoracic cage, transthoracic drainage has been considered the method of choice by most surgeons. The method employed in draining a subphrenic abscess, of course, depends largely upon the location of the abscess. Everything else being equal, drainage by the most direct route without contamination of a serous cavity will give the best results. The operations for subphrenic abscess may



GRAPH V.—Graphic representation of results obtained in collected and reported cases of subphrenic abscess, both operatively and non-operatively. At the right the results obtained by the various methods of approach in the collected and herein reported cases are graphically compared.

be divided into two large groups, *i.e.*, transthoracic and transabdominal. These, in turn, may be divided into transerous and extraserous approaches. A transthoracic extraserous approach is accomplished either by incising below the reflection of the pleura or by mobilization of the costophrenic angle of the pleura upward. The transabdominal extraserous approach is accomplished by incising over the abscess in extraperitoneal abscesses or by mobilizing the parietal peritoneum from the undersurface of the diaphragm until the abscess cavity is reached in suprahepatic infections. Similarly, intraperitoneal abscesses in which adhesions are present between the parietal peritoneum and abscess cavity can be drained to all intents and purposes extraserously, because if the incision is made through the area of adhesions no contamination of the uninvolved peritoneum will occur.

The transpleural method of drainage is too frequently employed. In the transpleural drainage an attempt is made to prevent contamination of

the pleural cavity by suturing the costal and diaphragmatic layers of pleura together, a procedure which was first suggested by Trendelenburg,[44] in 1883. That this procedure does not always protect the virgin pleural cavity against invasion is exemplified by Gatewood's[43] and our own statistics. In the only two of our cases (Cases XI and XX) in which suture of the costal and phrenic layers of the pleura were recorded at the time of operation, a fatal empyæma resulted. The results obtained in the collected series of subphrenic abscesses and in our own series demonstrate the danger of transpleural drainage. Of 305 collected cases drained transpleurally, 39.0 per cent. died. Sixteen of our own cases were drained transpleurally with a mortality rate of 50 per cent. (Chart VI.) Of the sixteen drained transpleurally, the free pleural cavity was opened seven times with a fatal outcome in six. (85 per cent.) Of the nine in which the free pleural cavity was recorded as not being opened, only two (22 per cent.) terminated fatally. Boeckel,[45] in 1889, suggested that transthoracic drainage could be safely performed in cases of subphrenic abscess without danger of injury to the pleura, because as a result of elevation of the diaphragm the costophrenic angle would be elevated above the line of incision. This conception is shared by many surgeons today and is undoubtedly one of the reasons why the transpleural operation is considered a safe one. However, Melnikoff[46, 47] has shown by his anatomical investigations that because of the fixation of the costophrenic reflection of the pleura to the ribs it is impossible for this portion of the pleura to become elevated, even though the elevation of the diaphragm may be extreme. One is not justified in assuming, therefore, that the danger of injuring the pleura is slight as a result of transthoracic drainage in those cases in which there is an elevation of the diaphragm. Melnikoff[46, 47] is of the opinion that in many of the cases in which it is thought that obliteration of the costophrenic angle has occurred as a result of adhesions the incision is actually made below the reflection of the pleura and, therefore, injury and contamination of the pleural cavity do not occur. Transthoracic extrapleural drainage may be accomplished, as suggested by Parijsky[48] by mobilization of the costophrenic angle upward. This procedure has been successfully employed by Melnikoff,[46, 47] Whipple,[10] and Elkin.[49] Brown[8] objects to this method, however, because he has found it difficult to mobilize the pleura due to its firm adherence to the diaphragm and chest-wall as a result of inflammatory reaction.

Similarly, transperitoneal drainage that permits contamination of uninvolved portions of the peritoneum is objectionable. Obviously, no such drainage should be attempted as illustrated by the reported statistics and also our own. In 307 collected cases in which transperitoneal drainage was used, but in many of which an uninvolved portion of peritoneal cavity was probably not traversed because of limiting adhesions, the combined mortality rate was 35.5 per cent. In our own cases twelve were drained transperitoneally with a mortality rate of 41.6 per cent. Of the eight which were drained through limiting adhesions and, therefore, without contaminating

uninvolved peritoneum, only one (12.5 per cent.) died. All the remaining four drained across uninvolved peritoneum died (100 per cent.). (Chart VII. Graph V.)

Ideally, therefore, drainage of subphrenic abscesses should consist of adequate evacuation of the abscess in such a way that contamination of the pleural and peritoneal cavities does not occur. We believe that this can be accomplished best by some type of extraperitoneal drainage, approaching the abscess cavity either from behind or from the front, according to the location of the abscess. In those cases in which the abscess is located in the right posterior superior space, which is the most frequent site involved both in the collected series (29 per cent.) and in our own series (60 per cent.) the "retroperitoneal operation" offers an ideal method of approach. It has the advantage that abscesses in the right posterior superior space and the frequently associated abscess in the right inferior space may be drained simultaneously through the same incision. It also has the distinct advantage that neither uninvolved portions of the pleural nor the peritoneal cavities are traversed or contaminated by the procedure. The technic of the "retroperitoneal operation" is as follows: Under paravertebral block analgesia and with the patient lying on the unaffected side on either a kidney rest or sandbag placed in the lumbar region so that a scoliosis of the lower dorsal and lumbar spine is produced, an incision is made over and parallel to the twelfth rib. (Fig. 3.) The entire twelfth rib is resected subperiosteally, care being taken not to injure the pleura which may be immediately beneath the rib. (Fig. 4.) The erector spinæ mass of muscles is retracted medially and a transverse incision is made at right angles to the spine across the bed of the resected rib at the level of the spinous process of the first lumbar vertebra. (Fig. 5.) It is extremely important that this incision through the bed of the rib be made transversely at this level and not parallel to the rib, because only in this way can one be sure that the costophrenic angle of the pleura will not be injured. Melnikoff[17] has shown that in 92 per cent. of the cadavers examined by him the costophrenic angle of the pleura on the right side either completely covered or touched the twelfth rib somewhere in its course. In 62 per cent. of his observations the pleura extended below the right twelfth rib. He further showed, however, that even though the relation of the costophrenic angle to the twelfth rib may vary considerably in different individuals, in no instance does the costophrenic angle extend as far caudally as the level of the spinous process of the first lumbar vertebra. Therefore, a transverse incision at the level of the spinous process of the first lumbar vertebra will invariably miss the pleura. (Fig. 6.) This incision passes through the bed of the twelfth rib and the attachment of the diaphragm, which in some instances is quite a definite structure, whereas in others it is represented only by a few frayed muscle fibres. After the diaphragm has been incised, the renal fascia is encountered. (Fig. 7.) This is continuous above and anteriorly with the posterior parietal peritoneum.

Summary of Cases in Present Report

CASE NO.	AGE	COLOR	SEX	ORIGINAL LESION	DIAPHRAM ELEVATED	SUBPHRENIC OPERATION	LOCATION OF ABSCESS	COMPLICATIONS	RESULT
1	28	C	M	9/8/32, Perforated peptic ulcer	+ 10/3/32	10/26/32, Retroperitoneal through bed of 12th rib	Rt. posterior superior	Bronchopleural fistula	Recovered
2	43	C	M	11/24/29, " " "	+ 12/14/29	12/14/29, Transpleural	Rt. anterior superior and rt. posterior superior	Pleura opened - empyema	Died
3	39	C	M	5/1/31, " " "	5/1/32	6/16/32, Tranperitoneal	Left anterior inferior		Recovered
4	40	W	M	2/4/29, " " "	+ 4/16/29	4/16/29, Retroperitoneal through bed of 12th rib	Rt. posterior superior		Recovered
5	39	C	M	5/3/27, Appendiceal abscess	+ 6/6/27	6/6/27, Transpleural	Rt. posterior superior		Recovered
6	16	C	F	8/28/22, Appendicitis	+ 9/12/22	9/13/22, Retroperitoneal below 12th rib	Retroperitoneal		Recovered
7	58	W	M	3/1/23, Perforated peptic ulcer	+ 4/6/23	4/9/23, Retroperitoneal through bed of 12th rib	Rt. posterior superior		Recovered
8	24	W	M	9/29/30, Cholecystitis		10/16/30, Transperitoneal	Right inferior		Recovered
9	9	W	M	1/6/33, Acute appendicitis	+ 1/16/33	1/17/33, Anterior extraperitoneal	Rt. anterior superior and rt. posterior superior		Recovered
10	30	C	M	6/1/24, Empyema	+ 11/15/24	11/19/24, Transpleural	Rt. posterior superior	Pneumonia	Recovered
11	26	C	F	5/27/24, Acute pelvic inflammatory disease		7/26/24, Transpleural		Pleura opened - empyema	Died
12	48	W	M	2/24/31, Perforated gastric carcinoma	2/24/31	2/26/31, Retroperitoneal	Rt. posterior superior and right inferior	Parotitis pneumonia	Died
13	34	C	M	5/12/26, Pneumonia	+ 6/29/26	7/10/26, Transpleural	Rt. posterior superior	Empyema	Died
14	23	W	F	9/1/26, Abortion	+ 10/13/27	10/17/27, Transpleural	Rt. posterior superior	Pleura opened - empyema	Died
15	39	W	F	8/1/23, Cholecystitis		8/26/23, Transpleural	Rt. posterior superior	Pleurisy	Recovered
16	40	C	M	2/27/32, Perforated peptic ulcer	+ 2/29/32	3/8/32, Retroperitoneal through bed of 12th rib	Rt. anterior superior		Recovered
17	18	W	M	6/15/32, Lobar pneumonia (left)		7/23/32, Rretroperitoneal below left 12th rib	Left retroperitoneal		Recovered
18	41	W	M	8/15/31, Operation for sinusitis	+ 10/14/31	11/8/31, Retroperitoneal below 12th rib	Left superior	Pleurisy	Recovered
19	32	C	M	8/25/27, Acute appendicitis	+ 1/30/28	2/2/28, Transpleural	Rt. anterior superior and rt. posterior superior	Empyema	Died
20	30	C	M	2/25/28, Cholecystitis	+ 4/27/28	5/9/28, Transpleural. Pleurae sutured	Rt. posterior superior	Pneumothorax and pleurisy	Died
21	35	W	M	6/20/28, Typhoid fever and pneumonia	+ 8/6/28	8/11/28 Retroperitoneal through bed of 12th rib	Rt. posterior superior	Pleurisy	Died
22	17	W	M	10/8/32, Acute appendicitis	+ 10/30/32	10/30/32, Retroperitoneal through bed of 12th rib	Rt. posterior superior		Recovered
23	18	W	M	2/1/33, Trauma of right lumbar region		3/10/33, Retroperitoneal through bed of 12th rib	Retroperitoneal		Recovered

24	50	C	M	8/1/26, Cholecystitis	+ 8/16/26	8/19/26,	Transperitoneal	Rt. inferior	Empyema	Recovered
25	24	C	M	1/22/31, Influenza	+ 3/26/31	4/2/31,	Retroperitoneal below 12th rib	Rt. posterior superior		Recovered
26	49	W	M	9/20/32, Perforated peptic ulcer	+ 10/1/32	10/6/32,	Retroperitoneal below left 12th rib	Left anterior inferior	Peritonitis	Died
27	24	W	M	7/12/25, Ruptured appendix	+ 8/13/25	8/13/25,	Transperitoneal	Rt. inferior		Died
28	17	W	F	11/6/33, Ruptured appendix	+ 12/12/33	12/24/33,	Retroperitoneal through bed of 12th rib	Rt. posterior superior		Recovered
29	30	C	M	10/27/26, Gunshot wound right costal margin		1/10/27,	Transperitoneal	Rt. inferior	Pneumonia	Recovered
30	18	C	M	6/9/31, Gunshot wound of duodenum		12/15/31,	Transperitoneal	Rt. inferior		Recovered
31	32	W	F	9/22/30, Acute appendicitis	+ 10/22/30	11/10/30,	Transpleural	Rt. posterior superior	Pneumothorax and empyema	Recovered
32	33	W	F	6/23/30, Acute appendicitis		6/30/30,	Transperitoneal	Rt. posterior superior		Died
33	54	W	M	10/27/26, Perforated peptic ulcer		2/27/28,	Transpleural	Rt. posterior superior	Pleura opened - empyema	Died
34	35	W	F	7/20/31, Acute appendicitis		7/20/31,	Transperitoneal	Rt. inferior and right posterior superior		Died
35	23	W	F	8/1/32, Cholecystectomy	+ 9/27/32	10/17/32,	Transperitoneal	Rt. inferior		Recovered
36	64	W	F	6/27/25, Ruptured appendix		8/27/25,	Retroperitoneal through bed of 12th rib	Rt. posterior superior		Recovered
37	12	W	M	9/7/24, Liver abscess ruptured	+ 9/9/24	9/25/24,	Transpleural	Rt. posterior superior		Recovered
38	12	W	M	8/9/30, Ruptured appendix		9/18/30,	Transpleural	Rt. posterior superior	Pleura opened - empyema	Died
39	46	W	M	10/25/32, Furunculosis	+ 11/10/32	11/11/32,	Extraperitoneal left	Left anterior inferior		Recovered
40	25	W	M	1/25/27, Perforated peptic ulcer	+ 2/7/27	2/7/27,	Transpleural	Rt. posterior superior	Pleura opened	Recovered
41	34	W	M	8/5/31, Cholecystostomy for hydrops	+ 8/14/31	8/14/31,	Transperitoneal	Rt. posterior superior and right inferior		Died
42	13	W	F	1/5/26, Influenza		1/12/26,	Transpleural	Rt. posterior superior	Pneumonia	Recovered
43	61	W	F	4/6/29, Cholecystectomy		6/5/29,	Transperitoneal	Rt. inferior		Recovered
44	26	W	M	Unknown	+ 3/23/28	3/28/28,	Transpleural	Rt. posterior superior	Pleurisy	Recovered
45	63	W	M	11/20/27, Perforated peptic ulcer		2/1/28,	Transperitoneal	Rt. posterior superior and right inferior		Died
46	53	W	F	4/20/26, Cholecystostomy		6/3/26,	Retroperitoneal below 12th rib	Retroperitoneal		Recovered
47	37	W	F	2/27/33, Acute appendicitis	+ 3/3/33	3/4/33,	Retroperitoneal through bed of 12th rib	Rt. posterior superior		Recovered
48	36	W	M	Peptic ulcer for eight years		4/13/33,	Retroperitoneal through bed of 12th rib	Rt. posterior superior		Recovered
49	40?	W	M	Unknown	+ 4/3/32	4/4/32,	Extraperitoneal left	Left anterior inferior		Recovered
50	39	W	M	4/25/33, Perforated peptic ulcer		4/30/33,	Retroperitoneal through bed of 12th rib	Rt. posterior superior		Recovered

CHART VII.—An analysis of personal cases.

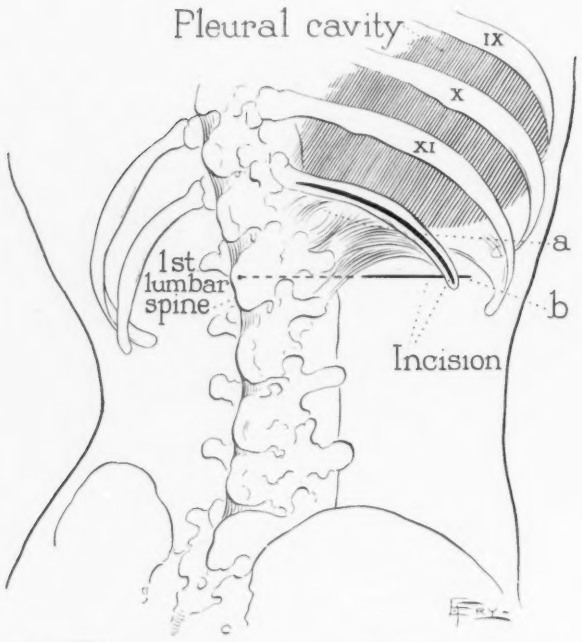


FIG. 3.—Diagrammatic drawing showing skin incision made over and parallel to the twelfth rib and transverse incision through the lumbar fascia and diaphragm at the level of the first lumbar spinous process.

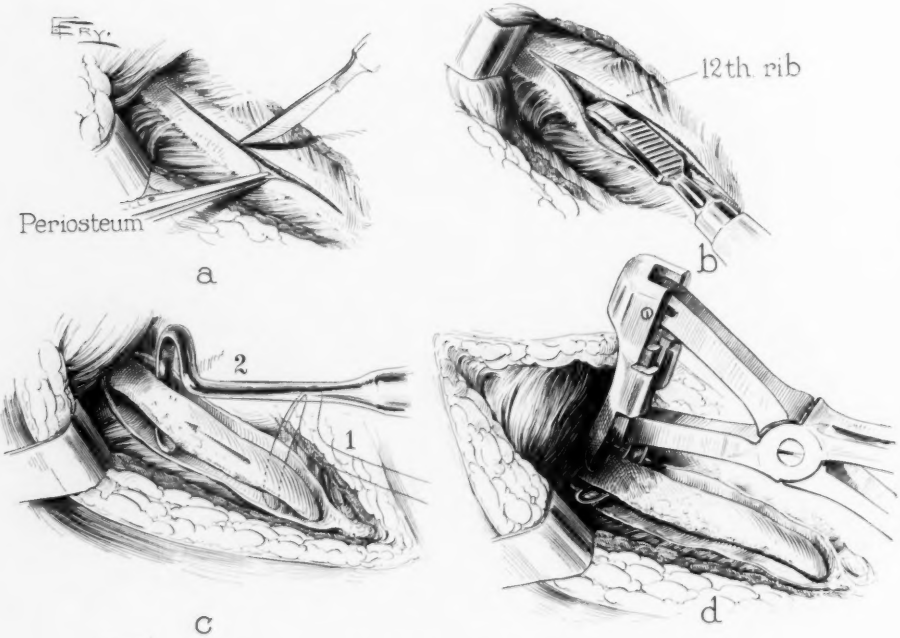


FIG. 4.—Subperiosteal resection of the twelfth rib throughout its entire length.

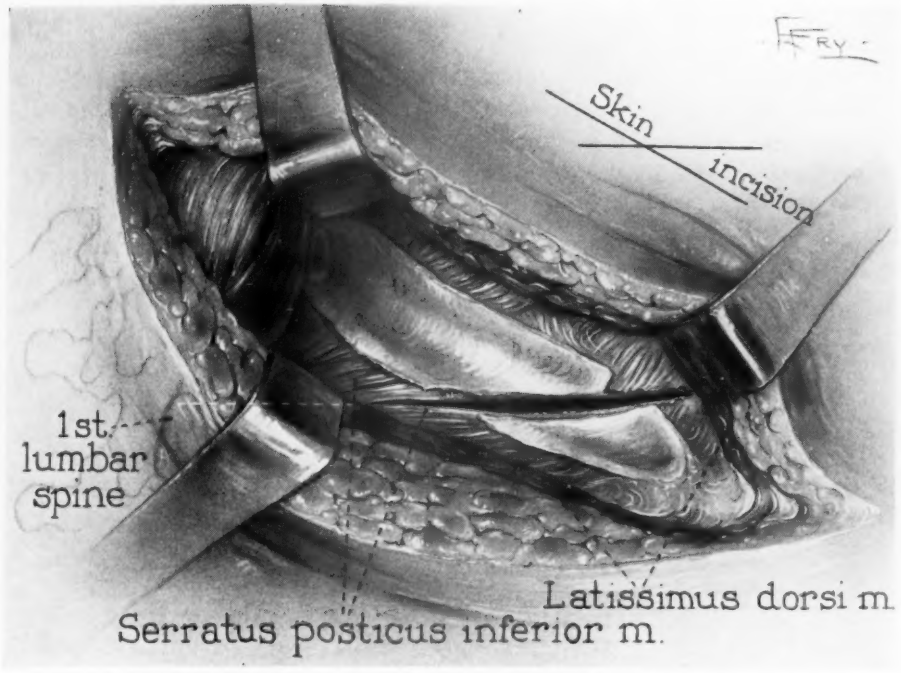


FIG. 5.—Drawing illustrating the transverse incision made through the resected bed of the twelfth rib and the serratus posterior inferior muscle at the level of the spinous process of the first lumbar vertebra. It is important that this incision does not parallel the original skin incision.

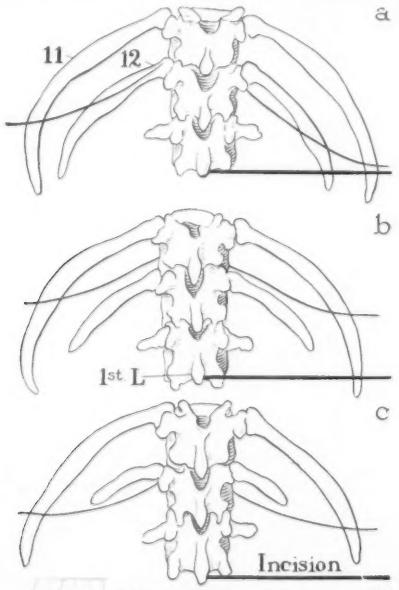


FIG. 6.—Diagrammatic drawing modified after Melnikoff, showing the variation in the relation of the costophrenic angle to the twelfth rib. It is evident that the transverse incision made at the level of the spinous process of the first lumbar vertebra will invariably miss the pleural reflection.

The kidney is displaced downward by means of the index finger and the infrahepatic space is palpated. If the symptoms and signs indicate and if at the time of operation an induration is found in the infrahepatic space, aspiration should be done in order to determine whether a suppurative process is present or not. In those cases in which a right posterior superior space abscess is suspected, the peritoneum on the undersurface of the diaphragm can be readily separated from the diaphragm by means of the finger. (Fig. 8.) This separation is readily executed in cadavers and in normal subjects and especially easily in patients with a subphrenic abscess. The inflammatory œdema which is invariably present greatly facilitates mobiliza-

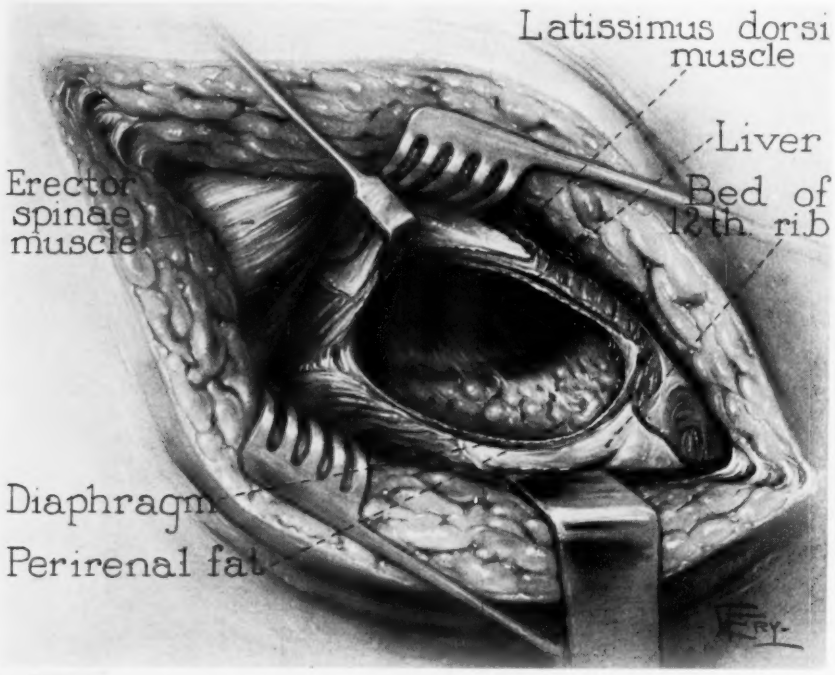


FIG. 7.—Drawing illustrating the operative wound following the transverse incision through the diaphragm at the level of the spinous process of the twelfth lumbar vertebra, exposing the perirenal fascia and the liver.

tion of the diaphragmatic peritoneum from the undersurface of the diaphragm. This separation may be carried upward as far as the dome of the liver and should be extended until the abscess is reached. By means of the mobilizing finger the abscess cavity is opened by plunging the finger through the abscess wall which is adherent to the mobilized parietal peritoneum. Large, soft, fenestrated rubber tubes are introduced into the abscess cavity and brought out through the wound. Through this incision adequate evacuation of abscesses located in the right posterior superior, right extraperitoneal, the right inferior, and even occasionally right anterior superior spaces may be accomplished without traversing or contaminating either the pleural or peritoneal cavities. The advantage of the retroperitoneal operation is exem-

plified by the results obtained in a total of thirty-one cases, nineteen of which were previously reported, of which three died, giving a mortality rate of 9.7 per cent. (Graph VI.) Two of the deaths were probably unavoidable as in each the patient died as the result of the original lesion, which in one was carcinoma of the stomach (Case XII) and in the other was typhoid fever. (Case XXI.)

Abscesses located in the right anterior superior, the right inferior, the left anterior inferior, and the left superior spaces can be drained extraperitoneally through the anterior abdominal wall. Obviously, however, the

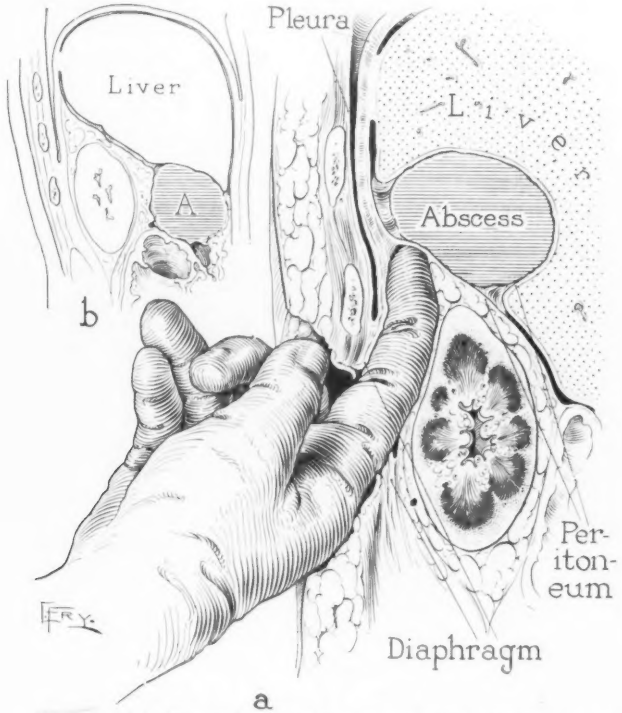


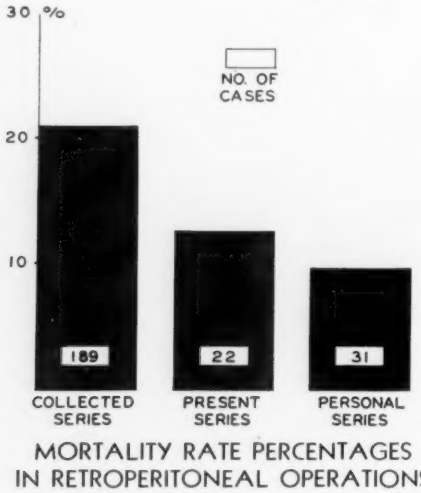
FIG. 8.—(a) Illustration showing the method of approach to an abscess in the right posterior superior space. By means of the finger the peritoneum is peeled from the undersurface of the diaphragm until the abscess cavity is reached. By plunging the finger through the abscess wall the abscess may be drained without contaminating the pleural or peritoneal cavity. Insert (b) shows location of a right inferior space abscess which may be drained simultaneously through the same incision.

anterior route of drainage would not be used in cases with right inferior space abscesses if retroperitoneal drainage is employed, because these abscesses can be drained satisfactorily by the retroperitoneal approach. In those cases (usually the right anterior superior and the left superior space infections) with abscesses above the liver, the suppurative process can be approached and drained extraperitoneally without contaminating uninvolved pleura or peritoneum by employing an approach suggested by Clairmont.[50] An incision just beneath the parallel to the costal margin is made through the flat abdominal muscles and transversalis fascia down to the anterior parietal

peritoneum. Similarly as in the retroperitoneal operation the parietal peritoneum is separated from the undersurface of the diaphragm by means of the index finger. (Fig. 9.) The peritoneum is mobilized upward until the abscess cavity is reached. The cavity is opened extraperitoneally through the abscess wall which is intimately adherent to the mobilized parietal peritoneum. Soft rubber drainage tubes or sheets of rubber tissue are introduced into the abscess cavity and brought out through the wound. In the present series there were three cases so treated, all of which recovered.

Summary.—(1) An analysis of 3,322 cases of subphrenic abscess collected from the literature and a presentation of fifty additional cases is made.

(2) The incidence of subphrenic infections is relatively high, but as



GRAPH VI.—Graphic representation of results obtained by retroperitoneal operation in the collected series, in the cases contained in the present report, and in personally operated cases.

most of these infections subside spontaneously and do not progress to suppuration, the incidence of subphrenic abscesses is much lower.

(3) The incidence of subphrenic abscesses in males is higher than in females, a ratio of three to one. There is no racial predisposition. Seventy per cent. of the cases in the present report were in the second to the fourth decades, inclusive.

(4) Subphrenic abscesses usually follow an intraperitoneal suppurative process. The most frequent antecedent conditions are perforated appendicitis and perforated lesions of the stomach and duodenum. In the collected and personally reported cases, appendicitis and perforated lesions of the stomach and duodenum were the originating focus in 59 per cent. and 54 per cent. of cases, respectively.

(5) The most frequent site of localization in subphrenic abscesses is the right posterior superior space, which was involved in 28.8 per cent. of the collected series and 60 per cent. of the cases in the present report.

(6) Diagnosis of subphrenic abscess is apt to be delayed because of the

inaccessible position of the abscess. Signs of persistent infection together with localized tenderness over the twelfth rib or along the costal margin suggest subdiaphragmatic infection. Diaphragmatic elevation and immobilization are of diagnostic importance.

(7) Subphrenic infections should be treated conservatively, because only approximately 30 per cent. progress to suppuration.

(8) Subphrenic abscesses should be drained surgically. In the collected

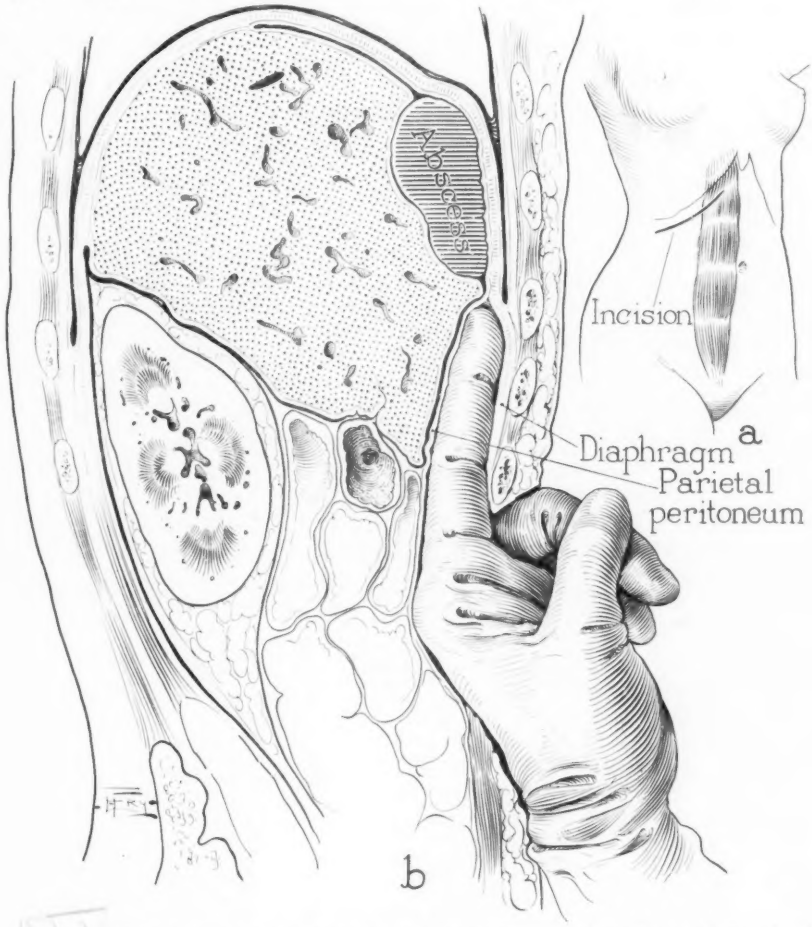


FIG. 9.—Illustration showing the method of draining extraperitoneally an abscess in the right anterior superior space. As shown in (a) an incision is made below and paralleling the right costal margin, passing through the flat abdominal muscles and the transversalis fascia. By means of the finger the parietal peritoneum is peeled from the undersurface of the diaphragm until the abscess cavity is reached. The abscess is then drained extraperitoneally without contaminating either the pleural or the peritoneal cavity.

cases treated non-operatively there was a mortality rate of 91.1 per cent. In the collected cases treated by drainage the mortality rate was 33.6 per cent. The mortality rate in the present series was 32 per cent.

(9) The mortality rate in the collected cases drained transpleurally was 39.0 per cent., as contrasted with a mortality rate of 21.1 per cent. in those cases drained without contaminating the pleura or peritoneum. The mor-

tality rate following transpleural drainage in the present series was 50 per cent.

(10) Of the cases drained transperitoneally the mortality rates in the collected and present series were 35.5 per cent. and 41.6 per cent., respectively.

(11) The mortality rates in cases drained retroperitoneally in the collected and present series were 21 per cent. and 13.6 per cent., respectively.

(12) In order to decrease the mortality in cases of subphrenic abscess, it is necessary that contamination of uninvolved portions of the pleura and peritoneum be avoided during drainage of the abscess. This can be accomplished best by draining the abscess extraperitoneally.

(13) The technic of the retroperitoneal operation is described and its value exemplified by the low mortality rate obtained following its use in thirty-one personally operated cases (9.7 per cent.).

We are grateful to those surgeons of the staffs of both the Charity Hospital and Touro Infirmary for some of the cases included in this report.

The authors are indebted to Dr. Ray Zeck for his preparation of a personally operated case and the privilege of including one of his cases in the present report.

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DISCUSSION.—DR. HOWARD LILIENTHAL (New York City) said there is one very important method of making a diagnosis of subphrenic abscess that has not been mentioned. He referred to the production of pneumoperitoneum, followed by X-ray examination with the patient in the erect position and also lying on the well, or supposedly well side. One gets an almost certain diagnosis not only of the abscess but of adhesions of the liver to the diaphragm. It is something that ought not to be omitted in suspected subphrenic abscess.

Concerning a point of technic in the operation, Dr. Charles A. Elsberg devised a posture very much like the knee-chest position, with a nice big pillow to compress the abdomen. We make an incision *upon* the ninth or tenth rib subperiosteally, very carefully, and push the pleura away. Remember the pleura is stripped easily posteriorly but it is difficult to push the pleura away anteriorly. Seldom one enters the thorax. If the pleura is accidentally violated, the tendency is for the air to remain away from the apex. Through a large incision exposure is good. The diaphragm is clearly visible and one can put a needle in and make an exact exploration. The abscess then can be freely opened. You can put your finger in or your whole hand and find secondary abscesses if these are present. He had opened liver abscesses also in this way. The reason why we go into the ninth or the tenth rib region is because in the usual subphrenic abscess the diaphragm is pushed far up, as you can see by the X-ray, in the presence of pneumoperitoneum. The incision must not be made too low. His preference is for the ninth rib.

DR. ARTHUR DEAN BEVAN (Chicago, Ill.) suggested that instead of resecting the twelfth rib, the incision be made and then the two gloved hands introduced and the structures stretched widely apart, and the neck of the twelfth rib either fractured or pulled loose from the ligaments which attach the twelfth rib to the transverse process. He had used that technic for a number of years. It is a very simple matter. After the transverse incision is made the two hands are introduced and then with some power the neck is either fractured or torn loose from the transverse process.

DR. ALTON OCHSNER (New Orleans, La.) said he had attempted on the cadaver to mobilize the pleura from the diaphragm as one mobilizes the peritoneum from the under surface of the diaphragm. It is quite evident, however, that at least in the cadaver the pleura is more intimately adherent than is the peritoneum to the diaphragm. Therefore, there is less danger, he believed, in opening an uninvolved serous cavity by the retroperitoneal approach.

In their particular series of cases in the transpleural group there were two in which at the time of the original operation the surgeon had sutured the diaphragmatic and thoracic layers of pleura together. In each one of these instances, even though suture had been done, a fatal empyæma resulted. That may have been poor technic and probably was, but still he believed that a transpleural approach will not give as good results as will an extraserous approach. The danger of opening the pleura can be obviated if one will use a transverse incision at the level of the spinous process of the first lumbar vertebra. Doherty and Rowlands suggested resecting only the distal half of the twelfth rib. He tried this in one case. In this particular instance the diaphragm was high. The abscess was high and he could not approach it until he resected all of the rib.

Frequently as in a kidney approach, unless one does resect the twelfth rib, one has difficulty in gaining access to the upper pole of the kidney. If one will strictly adhere to the transverse incision at the level of the spinous process of the first lumbar vertebra he was sure, based on Melnikoff's and his own anatomical investigations, that there is no danger of opening the pleura.

INJURY AS A CAUSATIVE FACTOR IN THE DEVELOPMENT OF MALIGNANT TUMORS*

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One of the most difficult questions that confront the industrial commissions today is: What part, if any, does injury play in the development of malignant tumors? While numerous papers have been written on the subject and it has formed the chief topic of discussion at national and international congresses, no definite conclusions have been reached; at least, none that has been universally accepted. The judges and commissioners who have listened to the opinions of medical experts have found it exceedingly difficult to balance these more or less conflicting opinions. Since the adoption of the Workman's Compensation Act, not only in this country but in Europe, there has come up for adjudication a rapidly increasing number of cases in which a claim for compensation has been made on the ground that a local injury was the exciting cause of a subsequently developing malignant tumor.

The most difficult thing in discussing any medical question, especially a medico-legal question, is for the physician or surgeon to preserve a judicial attitude and to bear in mind that the attitude of an advocate has no place in a scientific discussion. While this is an ideal we have not yet attained, it is a goal toward which we should aim.

During the last twenty or more years a great change has taken place in the attitude of the medical profession toward the question of trauma and its relation to malignant tumors. Many who formerly refused to admit a causal relationship have since become convinced by the steadily increasing evidence, too conclusive to admit of question. Furthermore, it has been definitely accepted by the courts and compensation bureaus not only in the United States but in most other countries as well.

In France, the whole question took on importance from a medico-legal standpoint as early as 1897. Then the first law was passed. This outlined certain conditions the fulfillment of which meant the establishment of a causal relationship between an antecedent local trauma and a subsequently developing tumor. In 1907, at the French Congress of Surgeons, Segond read his classical paper on the subject, in which he presented six conditions; which conditions or rules have been accepted not only by the courts and compensation bureaus of Europe but of America as well; they have been accepted by Ewing in his book on "Neoplastic Diseases."

These conditions imply the following: (a) The authenticity of the trauma. (b) Sufficient importance or severity of the trauma. (c) Reasonable evidence of the integrity of the part prior to the injury. (d) Correspondence of the

* Read before the American Surgical Association, May 10, 1933.

tumor to the site of the injury. (e) A date of appearance of the tumor not too remote from the time of the accident to be reasonably associated with it. (f) A diagnosis established by clinical and röntgenological evidence, supported when possible by microscopical examination.

The frequent association of trauma with malignant tumors impressed itself upon one of the writers (W. B. C.) as early as 1897,[1] when he presented a paper on the "Influence of Injury upon the Development of Sarcoma" before the New York Surgical Society. In this paper he analyzed 170 cases of sarcoma personally observed, forty-six of which gave a definite history of antecedent local injury. In 1910,[2] in a paper on "Injury as a Causative Factor in Cancer," he discussed the question more fully. At this time he reported 970 cases of sarcoma personally observed, in which there was a history of antecedent local trauma in 225, or 23 per cent.; and 250 cases of carcinoma, in which there was a history of injury in eighty-two cases, or 32.8 per cent. Of the latter group, 120 were breast carcinomas, of which fifty-two, or 42.33 per cent., gave a history of single antecedent trauma. A careful analysis of cases observed since that date shows about the same trauma-percentage: in a group of 360 cases of bone sarcoma personally observed from 1890 to 1926,[3] 181, or 50 per cent., gave a definite history of antecedent local injury. Since the publication of that paper we have observed 100 additional cases, making a total of 280 cases of sarcoma of the long bones associated with antecedent local trauma.

One of the reasons why the profession has been slow to accept the traumatic theory of tumors is because of the general skepticism on the part of the pathologists who, unable to find what they believe to be a clear or rational explanation of such causal relationship, have been inclined to attribute it to coincidence of a pre-existing tumor. We must bear in mind, however, that the pathologist does not come in direct contact with the patient, at least not in the early stages of tumor development. He has no first-hand information on which to base his opinion. On the other hand, the surgeon makes a physical examination. He learns on questioning the patient that the latter sustained an injury to a hitherto normal part, and that this exact part in the course of a few weeks or months has become the site of a malignant tumor. He cannot help but be impressed with the importance of the alleged injury and its possible relationship to the later-developing tumor.

In our opinion, the part that trauma plays in the etiology of malignant tumors is closely associated with the wider problem of the etiology of malignant tumors in general. We know that while a vast amount of study and research work has been done in an attempt to discover the cause of cancer, it still remains an unsolved problem. While the majority of pathologists at the present time undoubtedly believe cancer to be due to intrinsic causes (cellular theory), a considerable, and, we believe, increasing number, including surgeons who have had a large clinical experience with cancer in man, believe it is due to some extrinsic agent or microbic cause (parasitic theory).

In view of the increasing number of individuals who attribute their con-

dition, malignant tumor, to an antecedent local injury, it becomes more and more urgent for us to try to find out just what, if any, causal relationship does exist between the alleged trauma and the tumor. We cannot wait until the general problem of the etiology of cancer has been finally and convincingly settled.

Etiology of Malignant Tumors.—At present the profession at large, especially those engaged in cancer research, are divided into two main groups: *The first* and larger group maintains that malignant tumors are due primarily to some *intrinsic* cause, such as a congenital rest, or causes associated with the but little understood processes of cell development and cell restraint. Take, for example, a fracture. Here we find an immediate and very great out-pouring of new cells which form a callus or splint about the broken ends of the bone, and this callus quickly undergoes ossification with complete restoration of function. Why does this rapid multiplication of cells cease at the precise moment when no more are needed for the process of repair? We do not know, but we assume that there is some law called *growth-restraint*, which causes the process of proliferation to cease as soon as the damage has been repaired. Apparently Nature has some laws that govern the life and the death of cells. New cells are constantly being formed to take the place of old cells that have died or have been damaged by trauma, and when the damage has been repaired, the production of new cells ceases. In the case of a malignant tumor, however, the law of *growth-restraint* no longer functions. The multiplication of cells continues indefinitely, the new cells drawing their nutriment from the normal neighboring cells, thus weakening the individual until he finally dies of exhaustion or metastases.

In our opinion, there never has been any satisfactory explanation of the breaking down of the law of *growth-restraint*, nor of the difference in behavior of cells and tissues undergoing repair and those in the early stages of malignancy.

If we accept the theory of the intrinsic origin of cancer, the best explanation of the causal relationship of trauma is found in Ewing's book on "Neoplastic Diseases," third edition, p. 116. He states:

> "Important effects of trauma here are: (1) Solution of continuity, minute and gross; (2) separation of cell groups and tissue masses, as of skin, glands, bone; (3) necrosis of tissue; (4) confined hemorrhage requiring absorption or encapsulation; (5) accelerated regenerative processes with hyperemia, and new growth of specific cells, blood-vessels, and supporting tissue; (6) cicatrization.
>
> "Some of these conditions are well-known elements entering into the causation of tumors, and the failure of attempts to produce tumors by experimental trauma in given cases does not reduce their importance when associated with other necessary predisposing factors."

The second and smaller group believes that all malignant tumors are of parasitic origin due to some unknown intracellular microörganism. If we accept the parasitic origin of cancer, the explanation of trauma as a causative factor is simple, rational and logical: the trauma furnishes a favorable soil for the growth of the organism. We have merely to assume that an extrinsic

microörganism or infectious virus has in some way, through the circulation, gained access to the cell where it acts as an irritant, causing rapid proliferation and multiplication of the cells. All the new cells contain a similar microörganism. This process continues indefinitely until a malignant tumor is formed. The latter increases in size and finally causes the death of the individual.

On this assumption we can explain the development of metastatic tumors in other parts of the body: the tumor progresses in size; new blood-vessels and blood spaces are formed into which the tumor-cells frequently gain access and are carried to distant parts of the body, thus forming the nucleus of a metastatic tumor. That this latter tumor has the same histological features as the primary tumor is explained on the ground that the organism is an intracellular organism, and both the cell and the organism are transported through the circulation, thus producing a new tumor of the same type of cell as the original.

Theory Held by Dr. William B. Coley.—This theory, already described in detail in a paper read before the American College of Surgeons, in 1924, is briefly as follows: That there exists an unknown microörganism or several strains of this microörganism which is widely distributed throughout the world so that practically everybody is exposed to it, and yet it requires a favorable soil for its development into a malignant tumor. I do not think the question of "favorable soil" has ever received due recognition in discussions on the etiology of cancer. In a paper ("Some Thoughts on Cancer Control," American Journal of Cancer, February, 1928), I gave a more detailed account of this theory and cited the evidence in favor of it.

I suggested in 1924 that a similar explanation might be applied to the causation and development of malignant tumors. This would explain why everybody does not contract the disease—only those whose local resistance has been broken down by one of many factors, *e.g.*, local trauma or chronic irritation or some change in the chemistry of the body fluids possibly due to changes in diet or water.

Another condition that furnishes a close analogy is osteomyelitis. About one-third of the cases of acute osteomyelitis give a history of antecedent local trauma. Furthermore, it has been possible to produce the same result experimentally: by injecting a rabbit with cultures of *staphylococcus aureus*, no harm results; and yet if following the injection the rabbit receives a sharp blow on the tibia or some other bone, osteomyelitis quickly develops.

We may assume that malignant tumors in man are due to a microörganism that is latent in the circulation and which gives rise to symptoms of malignancy only after the normal resistance of the cells is broken down, in some instances by local trauma. The microörganism thus finds a suitable soil in the damaged cell, forming a symbiosis with the cell and causing a proliferation and multiplication resulting in a malignant tumor.

One of the strongest arguments in favor of the parasitic theory is the inhibitive and even curative action of the streptococcus of erysipelas upon various types of malignant tumors. As early as 1893, one of the writers

(W. B. C.) stated he could find no rational explanation of this action except on the assumption that malignant tumors themselves are caused by some type of microörganism.

Recently Bouveret,[4] of Lyon, France, published an important monograph on the "Pathogenesis of Cancer," in which he strongly maintains that cancer is an infectious disease due to some form of microörganism, probably to some strain of streptococcus of erysipelas. He bases his argument chiefly upon the inhibitive and curative effect of erysipelas upon malignant tumors, and believes that this action can be explained in no other way.

Another strong advocate of the parasitic theory is Gregoraci, of Naples.[5] He believes that the body-cells and tissues of every individual have either an inherited or an acquired defense against bacterial infection. He states that while microörganisms may occasionally cause an acute infectious process accompanied by fébrile reaction, they more often in an ultramicroscopic state install themselves in the intimate texture of the tissues or cells and await a suitable soil for further development. Having found a permanent habitat, either isolated or in association with other organisms, they proceed to draw their nourishment from the body cells.

A critical study of the whole question of trauma and tumors has recently been made by Dr. Leila Charlton Knox,[6] of St. Luke's Hospital, New York. Knox's main argument against accepting a single local trauma as a causative agent in cancer is based upon the fact that in a large amount of experimental work by Lubarsch,[7] Ribbert[8] and others, it was found impossible to produce a cancer by any form of local trauma.

The large number of clinical observations covering a period of nearly one hundred years which, in our opinion, furnish convincing evidence of a causal relationship between injury and malignant tumors, Knox brushes aside as of little or no value. She gives the impression of being in accord with Askanazy,[9] who stated that the literature dealing with the subject was only a "collection of anecdotes." We doubt very much if the majority of students of this question will agree that the clinical observations made by the leading surgeons and pathologists of the world, beginning with Virchow in his classical book entitled *Die Krankhaften Geschwülste* (1863), and including a long line of distinguished pathologists and clinicians can be completely ignored or justly classed as "anecdotes."

According to Knox, Segond[10] discussed the statistical collections of case reports of tumors of alleged traumatic origin, and doubted that they have any value, quoting Auguste Comté to the effect that they represent only "empiricism under a mathematical disguise, for the most extensive statistics when they are derived from a variety of sources often have less value than fifteen minutes of good observation."

Quoting further from Knox: "Ribbert,[8] who thought that all the statistical collections were without value, stated that well-studied single cases of this type might be more convincing than any heretofore published statistics."

With this statement of Ribbert we are in complete accord. The present paper is not a statistical collection of case reports gleaned from many hospitals, each one with its own system of history taking, but is a critical study of a large group of cases personally observed.

As we have frequently pointed out, the question is one in which the pathologist is less able to give a careful, judicial opinion than is the clinical surgeon, for the reason that he is always dealing with second-hand or hear-say evidence which in the court of law is regarded as of little or no value. Whereas the surgeon who sees the condition in the early stages and obtains a

first-hand account of the injury, if he is a practitioner of large experience and has a knowledge of human nature, is able to judge the credibility of the patient and to weigh the importance of the evidence. Scientific medicine has not infrequently made grave mistakes in ignoring the oft-repeated stories and beliefs of laymen simply because no satisfactory scientific explanation could be found for them. No better example of this can be found than in the discovery of the origin of tuberculosis. For hundreds of years the laity held a firm belief that tuberculosis was a contagious or infectious disease, but this the leading medical authorities denied. They based their opinion on innumerable statistics, chief of which were those of the Bromptom Home for Tuberculous patients showing that in thirty-five years not a single nurse or doctor had contracted the disease. In the following year Koch discovered the tubercle bacillus.

War Injuries.—Many writers who refuse to accept a causal relationship between injury and tumors base their contention on the almost complete absence of malignant tumors following war injuries. Shortly after the World War, Dr. John B. Walker (a Colonel in the American Army) sent us notes on fifty-six cases of sarcoma that were associated with recent fractures or gunshot wounds. These were as follows:

39 cases of sarcoma of the femur and tibia treated by amputation; 23 dead.
5 cases of sarcoma of the humerus treated by amputation; 2 dead
2 cases of sarcoma of the radius and ulna treated by amputation; 1 dead.

In a study of material from the Sanitary Reports of the Prussian Army from 1899 to 1907, Löwenstein[11] found 241 cases of cancer; of these, thirty-nine, or 16.5 per cent., were post-traumatic. In view of the regular physical examinations made in these cases, exact data as to the time and locality of the injury were available.

Löwenstein, whose evidence Knox regards as more reliable than that of Löwenthal, in his monograph on "Accident and Cancer," reported 271 critically chosen cases, of which 121 proved to be sarcoma.

Another argument frequently advanced by opponents of the *traumatic theory* is, that the number of cases of local trauma occurring in the daily routine of life is very large, while the number of cases of malignant tumor associated with antecedent local trauma is very small. The statistics of accident wards of large hospitals showing thousands of injury cases with but few if any tumor cases, are cited.

This whole argument when properly analyzed loses most if not all of its force. To begin with, no one believes that trauma alone can produce a malignant tumor. Hence the large number of injury cases with but few malignant tumors. To produce a tumor, other factors are required, *e.g.*, a predisposition on the part of the individual, inherited or acquired, resulting in tissues or cells of too low-resisting power to withstand the invasion of the microbic cause of cancer. In some individuals this resisting power is so low that no external cause is required for the development of the cancer; in

others, the natural resistance must first be lowered by some external force, such as local trauma or chronic irritation, before the disease can gain a foothold. Hence we should expect malignant tumors to develop not after all injuries but only when there is a co-existence of all the factors mentioned, which would account for the very small number of cases associated with antecedent local trauma. In poliomyelitis we find a close analogy. Here there is undoubtedly a microbic agent or virus, widely distributed, to which a great many are exposed, and yet, even in an epidemic, a comparatively few contract the disease. The explanation is, that nearly all adults and the great majority of children have a high degree of resistance or immunity to the organism. This resistance is either inherited, or acquired by having had an attack of the disease so light that it was never recognized.

As a matter of fact, we believe that the actual number of cases of malignant tumors in which there was some form of antecedent local trauma is considerably greater than the apparent number based on a study of hospital histories.

Two years ago, one of the writers was called upon in a single month to testify as expert in two cases of sarcoma that had recently been under his care at the Memorial Hospital or the Hospital for the Ruptured and Crippled. In one case no mention was made of antecedent local trauma, and in the other the house surgeon had stated that there was no history of trauma. In the latter case, the man had been thrown off a high ice wagon, striking his pectoral region upon a cobblestone pavement; a few weeks later a rapidly growing sarcoma developed at the exact site of the injury. In the former case, the patient had slipped while carrying a ladder under his arm, forcing the ladder against the soft tissues of his axilla and causing a bruise; shortly afterwards, a highly malignant tumor developed, at the exact site of the injury.

Fortunately, the writer had a complete personal history in these cases, with accurate description of the nature of the injury and the dates. A claim was brought against the insurance companies in both cases and full compensation was allowed.

If the errors in these histories had not been discovered, some later investigator of the question of trauma and cancer would have recorded both as cases without antecedent injury. If such events occur in hospitals in which a special effort has long been made to obtain exact information as to presence or absence of trauma in every malignant tumor, it is easy to believe that an even larger percentage of errors occur in the larger general hospitals.

Again, many pathologists base their opposition to a causal relationship between antecedent local trauma and the development of malignant tumors on the ground that so-called scientific or laboratory evidence of the integrity of the part at the time of the injury is lacking. Strictly, this would call for excision of tissue at the site and time of the trauma, for microscopical examination, which is manifestly impossible. On the other hand, the rules or conditions laid down by Segond call for no such laboratory proof but are satisfied with clinical and, when possible, röntgenological proof.

To cite a personal observation: A man was struck a severe blow on the occipital region by a heavy wooden packing case, producing a typical hematoma two and one-half inches in diameter over the occiput. Under two weeks' local treatment this diminished to about one-half its original size; it then began to increase. An operation was performed three weeks after the injury for a supposed hematoma but instead there was found an osteogenic sarcoma which had completely destroyed both tables of the skull over an area two inches in diameter extending to the dura. The diagnosis was confirmed by Doctor Ewing. Under irradiation and Coley's toxins, the disease apparently disappeared. At the end of seven and one half years, however, the patient is living with severe pain from radium osteitis, but no evidence of recurrence.* In this case there can be no reasonable doubt that the trauma was a causative factor in the development of the sarcoma. To suppose a pre-existing tumor without any physical signs or symptoms in such a location calls for a stretching of credulity beyond the ordinary limits.

In another case, a woman, while walking along the street, was struck a severe blow on the breast by a batted ball, causing a distinct bruise, ecchymosis, and severe pain. No tumor or swelling had been noticed in this region prior to the injury, and none was noticed immediately thereafter. However, two months later she developed a hard, rapidly growing lump at the exact site of the injury. This was pronounced malignant. A radical amputation was performed but the patient died a few months later. In this case, to assume the presence of a pre-existing, unrecognized carcinoma at the exact site of the injury, in our opinion, again calls for an unreasonable amount of credulity.

If such cases of clear-cut history of antecedent local trauma were rare or isolated the assumption of a pre-existing tumor might be warranted; but when we find the number increasing in direct proportion to the care with which the clinical histories are taken then we must look for some more rational or more probable explanation.

Wainwright,[12] of Scranton, Pa., in his paper on "Single Trauma, Carcinoma and Workmen's Compensation," maintains that: "If we will admit that the relationship has been a true one, even in one case, we must consequently admit that it may likewise be a possibility in any other case in which this relationship comes up for serious consideration."

According to Samuel Johnson, "Experience becomes the great test of truth and is perpetually contradicting the theories of men."

While Knox is but little impressed by Löwenthal's [13] paper on "The Traumatic Origin of Tumors," the latter after nearly forty years still remains one of the most exhaustive clinical studies of the subject that has ever been made. It is based on a careful analysis of 800 collected cases reported since 1870, with references to 360 cases of malignant disease of undoubted traumatic origin reported prior to 1863 and cited in Virchow's "Pathologic Tumors." The latter, however, are not included in the statistical presentation of the 800 cases. It is interesting to note that there were 137 cases of traumatic carcinoma of the female breast. Of the 316 cases of sarcoma reported, 167 were sarcomas of bone.

The time that elapsed between the trauma and the development of the tumor is stated in 190 cases, as follows: one month or less, 135 cases; one month to one year, thirty-three cases; upwards of one year, twenty-two cases. The longest interim stated was forty-nine years. In a few cases from fifteen

* Shortly after this was written, evidence of a local recurrence appeared and developed rapidly causing death in September, 1933, eight years after the treatment was begun.

to thirty-four years elapsed before the tumor was noticed. Löwenthal gives a brief history of all these 800 cases.

Carcinoma.—While a causal relationship between a single trauma and sarcoma had been more or less generally accepted, the English courts up to 1912 declined to accept any such causal relationship in cases of carcinoma. The first report of a legally established case of traumatic carcinoma of the breast we owe to W. Sampson Handley,[14] Hunterian Professor of the Royal College of Surgeons, London, who has long been regarded as the leading authority on cancer of the breast in Great Britain.

According to Handley, this patient, a woman, was referred to him on March 26, 1912. She stated that on November 3, 1911, she had fallen over a beam, striking on the left elbow and the left breast. The arm had to be kept in a sling for three weeks. About January 1, 1912, she first noticed a discharge from the left nipple, and shortly afterwards a small lump was seen in the left breast, which proved to be a large, malignant, rapidly growing duct carcinoma.

The case was tried and "the jury found for the plaintiff and awarded 200 pounds damages."

Janet Lane-Claypon, one of the foremost English authorities on cancer of the breast, who was selected by the British Ministry of Health to help compile the "Public Health and Medical Subjects" in 1924 and 1926, took charge of the investigation of cancer of the breast. She analyzed the histories of 508 cases of cancer of the breast selected from the leading hospital in London. In this number she found a definite history of antecedent local injury in 136 cases, or 26.77 per cent.

She divides the entire series into two groups, *e.g.*, *Group A*, in which there was a definite history of trauma followed by bruising; and *Group B*, in which there was a definite history of trauma without bruising, at least no statement of evidence of bruising was made in the history.

Group A contains forty-one cases. These she compares with a group of controls or 1,526 non-cancerous breast cases which showed only thirteen cases in which there was a history of bruise with cancer, the difference being 52.3 to 5.9 per cent. In Lane-Claypon's opinion the results of this study would lead one to believe that there was a definite association between injury and the subsequent development of cancer of the breast.

Group B, containing ninety-five cases, was compared with 1,526 controls or non-cancerous breast cases in which only eighteen gave a history of previous injury. A comparison of the two groups shows 62.6 per cent. of the positive cancer cases with a history of injury and 3.57 per cent. of the controls.

In a study of one hundred consecutive cases of carcinoma of the breast observed at the Presbyterian Hospital, McWilliams[15] found a history of antecedent local trauma in 44 per cent.

Our own personal series of 205 cases of carcinoma of the breast shows seventy cases in which there was a definite history of local trauma or in which the conditions laid down by Segond were practically fulfilled, seventy-one cases in which it was definitely stated that there had been no antecedent injury, and sixty-four cases in which no notation was made as to the presence or absence of trauma. Considering only the seventy cases in which there was a definite history of trauma and assuming that the sixty-four cases in which no statement was made were not associated with injury, we have 34.1

per cent. of the entire series in which there was a history of antecedent local trauma.

The rôle played by trauma in the development of metastases in latent carcinoma has been discussed by Firket of the University of Liege, who reports an unusual case, one of the few on record, that illustrates this point. His patient, a woman aged forty-five years, had a carcinoma of the rectum for which he performed a radical Kraske operation in the spring of 1912. The patient made a complete recovery and remained in good health without any symptoms of disease in any other part of the body until May, 1916, when she let fall on her foot a heavy earthen bowl. While there was no open wound, a very definite, painful contusion developed almost immediately afterwards. The severe pain never subsided, and two months later, a definitely outlined, hard, non-fluctuating tumor could be made out. *Röntgen-ray diagnosis.*—Tubercular osteitis. The tumor increased in size rapidly and became ulcerated. Three months after the accident the foot had grown to an enormous size and was very painful. An amputation was performed, and on microscopical examination the tumor proved to be a cylindrical-cell carcinoma, the same type as the carcinoma of the rectum.

The history in this case is so precise that it would seem impossible to explain away the causative influence of the trauma on the supposition that there was a pre-existing tumor at the site of the injury. This adds one more to the rapidly increasing list of cases which, following the suggestion of the English surgeons, may be classed as *acute traumatic malignancy.*

The foregoing case closely resembles one reported by one of us (W.B.C.) in 1912, except for the important fact that in our case there was no long "period of latency" between the development of the primary tumor and the metastatic tumor; as a matter of fact, the latter was discovered before the primary tumor had been recognized by any one. The patient, a boy aged six years, was admitted to the Hospital for Ruptured and Crippled on February 20, 1910, as an ordinary case of left inguinal hernia. Operation disclosed an uncomplicated left inguinal hernia which was closed by the Bassini method. The wound healed by primary union and the patient was discharged at the end of three weeks. Seven weeks later he was readmitted with a large swelling in the inguinal region directly under the hernial incision, extending from the anterior superior spine to the upper scrotum, not involving the testicle. The swelling was entirely painless. It was first noticed the week previously by the family physician who had been called in for what was supposed to be an ulcerated tooth, and who, on learning that the patient had been operated upon for a hernia, of his own accord examined the scar and found the swelling described.

On readmission examination showed a fusiform, sausage-shaped swelling, beneath the skin, extending the entire length of the hernial incision. The first impression was that we were dealing with some inflammatory exudate, but there was no fluctuation nor tenderness on pressure, no pain, and no temperature. The skin was normal in appearance. In consistence the swelling was firm but not hard, and from the clinical features, particularly from the "feel" of it, a diagnosis of sarcoma was made by one of us (W.B.C.).

On further questioning it was learned that the patient had had two teeth extracted the week previously because of ulceration. No one had suggested that the condition of the mouth might be due to a neoplasm and not inflammation. On carefully examining the jaw it seemed to me (W.B.C.) quite evident that we were dealing with a malignant, not an inflammatory, condition, and that in all probability this malignant tumor of the jaw antedated the tumor of the groin and had probably been present at the time of the operation although not sufficiently advanced to give rise to any symptoms. Sections from the tumor of the jaw and from the groin were examined by Doctor Ewing, who pronounced both to be round-cell sarcoma. The tumor proved to be a highly malignant

one. After a very brief course of toxin treatment, or two weeks after his entry, the patient was removed from the hospital because of family troubles. Even in this short time the disease had advanced with great rapidity, especially the tumor of the jaw, which had extended up in the orbit almost completely closing the eye. At the same time the glands of the groin and iliac fossa had become involved. The patient died three months from the time the jaw tumor was discovered.

While this is apparently an unique case little if any reference to it has been made in any of the literature on the subject. The most rational explanation of this case is that some of the cells of the unrecognized tumor of the jaw, carrying with them in their nuclei the unknown microbic agent, entered the circulation, but caused no metastases until the local resisting power of the normal body cells was lowered by the trauma of the hernia operation a few weeks before. As a result of this trauma, the exudate and the slight hæmorrhage associated with the operation, furnished just the soil suitable for the development of the organism, hence the rapid development of the metastatic tumors.

FIG. 1.—Carcinoma of tissues over sternum developing two weeks after severe blow. (See Case I.)

The following case we believe represents the most convincing example of *acute traumatic malignancy* in carcinoma that has ever been reported:

CASE I.—A. K., male, aged fifty-nine years, who, while performing his duties as a watchman on November 6, 1931, fell downstairs, receiving a severe blow over the upper part of his sternum from a metal clock that he was carrying. About two weeks later (Fig. 1) a small purplish area appeared in the upper portion of the sternum at the exact site of the injury. This soon began to increase in size and became protuberant. It grew very rapidly, and in the latter part of December, 1931, the patient was admitted to Fordham Hospital. An aspiration needle was introduced into the tumor several times in the belief that it might be an abscess. On January 13, 1932, the patient was referred to Dr. William B. Coley.

Physical examination at this time showed a mass the size of half an orange situated over the upper portion of the sternum, extending above the sternum, and infringing on

the neck over the thyroid gland. It was about three and one-half inches in diameter, and elevated two inches above the normal surface of the sternum. It was purplish-red in color. There were several areas slightly ulcerated and discharging at the site of the previous aspirations. The tumor was firm in consistence over the larger portion—in fact, it was markedly indurated and characteristic of a carcinoma rather than a sarcoma. It was ulcerated over the most protuberant portion, and softer, almost semi-fluctuating over some areas. There were a number of outlying glands along the cervical region, and marked enlargement of the glands in the left axilla. Some of the latter had reached the size of an English walnut, were hard, and typically carcinomatous in character.

The patient was admitted to the Memorial Hospital January 15, 1932, where an aspiration biopsy was performed, and the clinical diagnosis of carcinoma was confirmed microscopically by Doctor Stewart. The condition was so far advanced that it was regarded as hopeless by all who saw the patient. Röntgen therapy had no effect in checking the progress of the disease. Pulmonary metastasis developed in a few weeks and the patient died on February 18, 1932. The entire duration of the disease from the time of the injury until death was only a little over three months.

In this case it was difficult to reach any other conclusion than that the single local trauma was the exciting cause of the disease.*

In another case, a man while working in a machine shop was struck over the malar bone by a piece of metal. A swelling appeared almost immediately; this never subsided but increased rapidly in size. At the time of the patient's admission to the Memorial Hospital, it had reached the size of a goose egg. While it appeared more like an inflammatory condition, operation by one of us (W.B.C.) revealed a malignant tumor. This diagnosis was confirmed by Doctor Ewing. The tumor, which had been removed as completely as possible, recurred promptly, and the patient died within three months.

These two cases, in our opinion, furnish convincing evidence of the occurrence of *acute traumatic malignancy* in carcinoma as well as in sarcoma.

The following case is another interesting and convincing example of *acute traumatic malignancy*. I am indebted to Dr. William L. Watson for the history.

Case II.—D. B., female, aged twenty-one months, on February 21, 1933, while playing on the floor, crawled under the gas range. She had difficulty in getting out, and becoming frightened, struck the top of her head against the range. The mother noticed a small abrasion on the scalp, but no bleeding. On the following day while bathing the child she found a small lump at the side of the injury. Five days later, no improvement being noticeable, she took the child to Dr. J. Edgar, of Jersey City. He regarded the lesion as a hematoma and prescribed local applications and gentle massage. He examined the patient again one week later, when the hematoma showed some evidence of softening, but did not advise any treatment. April 6, or forty-three days after the injury, the mother again consulted the doctor, calling his attention to the change in color of the swelling (it had become dark purplish) and to apparent increase in size. He, in consultation with another physician, then made a diagnosis of sarcoma, and referred the patient to the Memorial Hospital April 11, 1933, where she was placed on the service of Doctor Watson.

Examination on admission showed a firm, pale, purplish tumor mass measuring 6 by 5½ by 2½ centimeters, situated in the vortex of the scalp, and involving the skin. Numerous firm, hard subcutaneous nodules ranging in size from 2½ to 3 centimeters were scattered throughout the occipital scalp and neck.

Provisional diagnosis. (Dr. F. Stewart.) Endothelioma of scalp with metastases to both sides of the neck.

Treatment. Low-voltage X-rays.

* At the trial the Insurance Company made no attempt to deny a causal relationship, and the referee awarded full compensation.

By April 26 the primary tumor had apparently disappeared and the metastatic masses had practically vanished. A röentgenogram taken at this time showed no definite evidence of bone involvement. An aspiration biopsy was performed, and the following microscopical diagnosis made: unclassified round-cell malignant tumor, possibly endothelial myeloma.

In the foregoing case we believe it would be difficult if not impossible to apply Knox's method of reasoning, *i.e.*, that there must have been a pre-existing tumor at the site of the trauma. Here we have a young child with very little hair on her head, who was bathed daily by her mother. The latter is positive that there was no lump or swelling of any kind prior to the injury. The small lump or hematoma did not develop until the day after the injury and then, instead of subsiding as an ordinary hematoma would be expected to do, it slowly increased in size. Forty-three days later it had reached the size shown in the accompanying illustration (Fig. 2) and had metastasized.

FIG. 2.—Highly malignant metastasizing tumor developing a few days after local trauma. Patient now has general metastases. (See Case II.)

Neither do we believe that Knox's method of reasoning could be applied in our Case I, A. K., which in many ways is similar to the preceding case. Here there is every reason to believe that the tissues over the upper sternum were normal until the time of the injury; there was no evidence whatever of a pre-existing tumor. The swelling did not develop until nearly two weeks after the injury, and was then regarded as either an abscess or a hematoma. In this case, as in the preceding, extensive metastases to the glands of the neck developed but in an even shorter period, *i.e.*, two weeks after the beginning of the tumor and four weeks after the injury.

The question, why a single trauma is capable of changing a pre-existing benign tumor of long standing into a malignant tumor, is one that has been

occasionally referred to in monographs and text-books, but we believe the following case is the only one which has come before the courts for adjudication. According to Ewing, a "pre-cancerous condition may be precipitated into a malignant process by injury. Examples are wounds of a psoriatic tongue by the teeth, injuries of the breast altered by chronic mastitis, and incomplete surgical removal of indolent ulcers, mucous polyps, fistulous tracts, and benign tumors."

In the following case one of the writers (W. B. C.) testified as a medical expert in July, 1932:

CASE III.—Multiple malignant tumor apparently caused by single local trauma.

W. J. N., male, aged sixty-two years, August 29, 1931, was injured in an automobile accident. The sedan in which he was riding was crashed into by a bus, the impact being of such force as to push the sedan forward, up a small embankment, and over on its side. The plaintiff, who was sitting beside the driver, was thrown to the left, striking his leg against the gear shift and emergency brake. He suffered a dislocation of the left shoulder, and felt sore and bruised all over, especially over a small tumor, about the size of a hazelnut, situated in the middle of the right leg, which had existed without any appreciable increase in size for fifteen or twenty years. The family physician, Doctor Bloom, who was called in the same evening, examined the shoulder only, no other part of the body. It was not until five or six weeks later that the plaintiff showed his leg to the doctor. He stated that about three or four weeks after the accident he noticed an area of inflammation on the right leg about a quarter or three-eighths of an inch away from the lump. Six or possibly eight weeks after the injury he began to feel intense pain in the lump on his leg. Three months after the accident the lump had grown to the size of a very large hen's egg. This same lump had been noticed by Doctor Bloom three or four years prior to the accident, and in the belief that it was a sebaceous cyst he considered the possibility of a surgical removal. It was normal in color, of fair consistence, and freely movable under the skin. It had remained practically stationary in size during the period of his observation, but examination five or six weeks after the accident showed it to have markedly increased in size. On his advice, it was removed on December 15, 1931.

Microscopical Diagnosis.—Mixed spindle- and giant-cell sarcoma, malignant.

After three or four weeks' X-ray treatment, a second operation was performed in January, 1932, and in April, 1932, the leg was amputated about six inches above the knee.

The controversial question in this case was, whether or not the injury sustained by the plaintiff in the accident was the cause of the sarcoma which developed on his leg and required its amputation. The plaintiff maintained that the growth on his leg had been there for fifteen years, a benign, quiescent nodule, of firm consistence, movable under the skin, and that in all probability it would have remained as such throughout his lifetime but for the intervention of the accident. On the other hand, the defendant claimed that the growth was at the time of the accident and always had been a neurogenic sarcoma, such as it was found to be when the first operation was performed, and that it was not caused by the injury received in the accident.

An eminent pathologist who testified as an expert in behalf of the plaintiff expressed the opinion that the injury or blow received on August 29, 1931, was competent to stir up and make malignant the quiescent nodule on the man's shin. He stated that he had seen two cases of neurogenic sarcoma in which the growth had followed immediately after an injury.

One of the present writers (Dr. William B. Coley) also testified as an expert in

behalf of the plaintiff. In his opinion the blow on the leg on August 29, 1931, was a competent producing cause of the sarcoma or malignant condition which was found some three or four months later. He cited cases of neurogenic sarcoma coming under his own observation, in which a malignant tumor had developed shortly after an injury and at the exact site of the injury, the diagnosis being confirmed by microscopical examination and the fatal termination of the disease. He also cited two cases of quiescent pigmented moles which shortly after a local trauma became rapidly growing malignant melanomas.

Another eminent pathologist who testified as an expert in behalf of the defendant expressed the opinion that the plaintiff's condition was the natural history of a neurogenic sarcoma. The latter, he stated, has its own mode of growth and behaves as it does for causes that are inherent in the original tumor. He admitted the possibility of an adequate trauma causing a quiescent tumor to growth more rapidly.

Excerpts from Ewing's book on "Neoplastic Diseases" were read, as follows: "Mechanical trauma is an important factor in the causation of tumors. . . . The predisposing factors take many forms; there may be a benign or a minute malignant tumor in the tissue before the injury.

"Second. The precancerous condition may be precipitated into a malignant process by injury. Examples are wounds of a psoriatic tongue by the teeth, injuries of the breast altered by chronic mastitis, and incomplete surgical removal of indolent ulcers, mucous polyps, fistulous tracts and benign tumors."

The foregoing is sufficient to give the facts of the case and to show the conflicting opinions expressed by the experts. After nearly a week of argument the case was submitted to a jury who rendered a verdict of forty thousand dollars in favor of the plaintiff. This amount was reduced to twenty-five thousand dollars by the court. An appeal from this decision was made and carried before the Appellate Division of the Supreme Court on April 6, 1933. The decision was rendered July 14, 1933, affirming the first decision.

Intrathoracic or Intra-abdominal Tumors.—While it is now very generally admitted that a single local trauma may be an exciting factor in the development of a malignant tumor at or near the external surface of the body, very few are willing to admit such a causal relationship in cases of intrathoracic or intra-abdominal tumor. Knox,[6] in her review on trauma and tumors, stated "that serious injuries to the chest are so frequent and pulmonary tumors so rare that, statistically, a causal relationship is not even suggested." On the other hand, Aufrecht[16] regarded severe trauma which "does not produce laceration of the pulmonary tissue, but only molecular disturbances of an unknown character," as an important immediate cause of carcinoma of the lungs. He cited four cases personally observed in which the pulmonary carcinoma was preceded by severe trauma. These cases were regarded as of sufficient importance for Ewing to refer to them in his book on Neoplastic Diseases (*loc. cit.*).

In a recently reported case of primary carcinoma of the lung, Wells and Cannon[17] offer what we believe to be most convincing proof of a causal relationship between the carcinoma and the trauma which preceded it. This case is briefly as follows:

Male, aged fifty years, had always been in good health until September 1, 1926, when he was knocked down by an automobile. Severe pain in the chest followed. A röntgenogram taken on the next day showed a fracture of the left third, fourth and fifth

ribs in the mid-axillary line. There was also distinct evidence of traumatic injury to the lung, namely, hemoptysis and a subcutaneous emphysema extending over the entire body. No evidence of any neoplasm in the lung was revealed by the röntgenogram. The patient made an uneventful recovery and seemed to be in good health until the following August, when he complained of pain in the left side of the chest. A cough developed, and while symptoms suggestive of pulmonary tuberculosis appeared, no tubercle bacilli could be found in the sputum. Röntgenograms taken at this time revealed evidence of cancer in the left upper lobe of the lung. The clinical course was steadily downward ending in death on August 17, 1928, or one year after the development of symptoms and barely two years after the injury to the left lung.

A *post-mortem examination* made by Doctor Paul R. Cannon revealed the presence of a primary carcinoma of the upper lobe of the left lung, with metastases to the mediastinal and left supraclavicular lymph-nodes, in the retroperitoneal peri-aortic lymph-nodes as far down as the bifurcation in the right suprarenal gland and both kidneys. There was a thickening and an irregularity in the third, fourth and fifth left ribs in their middle thirds from the healed fractures There were no tumor nodules in the right lung.

Histological Examination showed the tumor to be composed of cells which generally appeared elongated, consisting chiefly of nucleus with little cytoplasm, thus much resembling sarcoma cells, but they tended to form alveoli, did not secrete collagen, and often exhibited a palisade arrangement. In no place did the cells exhibit a characteristic epithelial structure, nor did they form tubular structures, secrete mucin, or undergo keratinization. In other words, the structure was that of the type of lung tumors that has often been described in the earlier literature as sarcoma and later as mesothelioma, and which has been interpreted by some as a tumor arising from the flat epithelium of the alveoli.

According to Wells and Cannon, this case "seems to present as nearly completely satisfactory evidence as one can hope to secure of the development of a primary carcinoma of the lung as a direct result of a single traumatism to the lung tissue. Röntgen plates of the chest made immediately after the injury show that at this time there was no evidence of a carcinoma of the lungs demonstrable by this means. There is conclusive evidence of traumatism to the lung (hemoptysis and severe subcutaneous emphysema). The interval between the time at which the traumatism was received and the appearance of symptoms of the cancer of the lung (eleven months) is entirely in harmony with the assumption that the neoplastic growth was caused by the traumatism of the lung, and the duration of life after this time (twelve months) is in keeping with the rate of growth to be expected from a tumor reaching the observed state in the first eleven months after the traumatism. . .

"Of course, it is not possible to say that there was not already a carcinoma, too small to be detected in the Röntgen film, growing in the part of the lung that was traumatized at the time of the injury. But in view of the extreme infrequency of primary carcinoma of the lung arising in the periphery of the upper lobe, to support such an explanation of this particular case requires a stretching of 'the long arm of coincidence' to the vanishing point."

Personally, one of the writers has observed only one case of intrathoracic tumor in which he was fully convinced that the single trauma experienced was the exciting causative factor of the development of the tumor. This case later became one of the most important medico-legal cases that has ever come before compensation boards in this country. It was reported by Doctor Coley at a meeting of the New York and New England Association of Railway surgeons. In certain respects it closely resembles the case reported by Wells and Cannon.

CASE IV.—L. D., aged thirty-six, weight 200 pounds, had always been well until July 30, 1921, when while working, he was caught between scaffold and stone coping of roof, receiving a severe bruise over the ninth, tenth, and eleventh ribs on left side. Felt very sharp pain and great difficulty in breathing, which continued so that he found it impossible to sleep while lying down. Three days after injury, physician was called who found swelling, ecchymosis and tenderness at site of injury. He tried to do light work for two or three weeks, but gradually became worse and coughed up blood, so gave up working. No X-ray taken at time of accident, but six weeks after injury, X-rays showed what was taken to be exudate in pleural cavity at site of injury, and diagnosis of traumatic pleurisy was made. Patient grew rapidly worse and died, January, 1922. Full course of disease less than five months. Autopsy showed malignant tumor involving ninth, tenth, and eleventh ribs at exact site of injury, also tumor of left lung, tumor of right lung and liver. Microscopical examination by Doctor Ewing: epidermoid carcinoma.

During period of five years case came before four referees. Attorney for plaintiff (widow), claimed injury was competent producing cause of death. Attorney for defendant (Insurance Company, carrier), claimed cancer was primary in lung for considerable period before injury, and that injury had no causal relationship with tumor, nor did it in any way accelerate condition.

First trial: Verdict in favor of defendant.

Second trial: Lasted a year, due to various postponements; referee went out of office.

Third trial: Case reheard from beginning. Expert for defendant, pathologist of great experience, testified that histological type of tumor, epidermoid carcinoma, ruled out possibility of its originating in ribs or tissues about site of injury. One of writers (W. B. C.), testifying as expert for plaintiff, expressed opinion that all clinical facts of case, pointed to tumor being primary at site of injury, not at root of lung; he believed tumor at root of lung to be metastatic from tumor at site of injury. This clinical evidence, he believed, outweighted that based solely on the histological type of tumor, for the reason that tumors of the lung are recognized as extremely difficult to classify exactly, some pathologists calling a case epidermoid carcinoma and other endothelioma. Verdict of third referee in favor of defendant.

Fourth trial: Plea to have case reopened granted by Hon. Frances Perkins, now U. S. Secretary of Labor, who had succeeded referee who made last decision. Case again heard in fall of 1927, and in July, 1928, final verdict was rendered by Commissioner Perkins in favor of plaintiff, reversing the previous decisions. A copy of her decision may be of interest:

"Because the question of fact in this case was considered to be extremely close, the record has been personally reviewed by four members of the Board, each reading independently and writing a memorandum of decision without conference with the others. The only question involved is that of causal relation between the accidental injury and the death of L. D.

"Three members of the Board have found causal relationship to be established and one considers the weight of medical evidence to be against such a finding. The Board, therefore, finds that L. D. sustained a crushing injury to his chest wall on July 30, 1921; that the injury was serious is shown by the fact that he had difficulty in breathing and continuous pain in his chest for many weeks. In September, 1921, he had a hemorrhage and spat blood and pus. The pus when analyzed showed streptococcus and staphylococcus.

"There can be no doubt of the inflammatory condition or that it resulted from the accident. This indicates serious injury to the pleura. The case was diagnosed by his physician as traumatic pleurisy. He grew constantly worse, was in a hospital for sev-

eral months, treated for pleurisy, broncho-pneumonia, with some physicians suspecting tuberculosis. He had been a man in exceptionally good health prior to the accident but he declined rapidly. On January 11, 1922, he died, still a puzzle to the hospital physicians. An autopsy was performed and an epidermoid carcinoma was found to have involved the lungs, ribs, kidney and liver. This carcinoma is stated to be the cause of death.

"The contest has been as to whether there was a causal relationship between the injury and this cancerous growth which progressed so far as to cause death. The testimony on this point has been difficult to follow because obscured at times by antagonisms and by arguments and confused by objections, interruptions and comments by counsel for both sides to a degree at least unusual in this jurisdiction. His family physician who treated him throughout as of the opinion that death was the result of the accident. There is also other expert medical testimony to this effect.

"After long and careful consideration, the Industrial Board finds that the death resulted naturally and inevitably from the accidental injury."

In reporting this case (see 1929 Year Book, New York and New England Association of Railway Surgeons) Doctor Coley cited two other cases in which very similar verdicts had been rendered by the Supreme Court.* In the first, the referee ruled that "compensation is payable where death occurs within 300 weeks of the time of the accident, provided the testimony shows it was caused by the injury, or, by reason thereof, an incipient condition was hastened to development, ending in the loss."

In the second case it was ruled that "claimants'" right to recover compensation is controlled by section 2 subdivision (d), of the Workman's Compensation Act (Law 1918, c. 400), which is as follows:

"'Injury' and 'personal injury' shall mean only injury by accident arising out of and in the course of the employment and shall not include a disease in any form, except where it results naturally and unavoidably from the accident. . . .

"It is conceded that if there is evidence to sustain the finding that the sarcoma resulted from the alleged injury, or if it was at the time of the accident in a quiescent state and the accident aggravated it and hastened the employee's death, then the requirements of the above-quoted section are met and the present claimants are entitled to compensation."

The referee continued as follows:

"Whatever view we take of the medical opinion, they are frankly and at best but theories, but taking them as they are in connection with the facts heretofore narrated and taking a common-sense, practical view, as courts and commissions must take of the ordinary happenings of life boiled down to its last analysis, the medical theory is that there is a relationship between the receipt of injury and orgin of sarcoma, and that the degree of injury plays no important part. With this in mind we find a perfectly healthy, strong man, who has never lost any time from work or complained of any illness, suffers an injury and from that time on is incapacitated, grows worse and worse, sarcoma develops at the point of injury, from which he dies. The lay mind, under such

* Smith *vs.* Primrose Tapestry Co., 131 Atl. Rep. 703 (285 Pa. 145), decided by the Supreme Court, Pennsylvania, January 4, 1926. Winchester Milling Corporation *et al.* V. Sencindiver *et al.* 138 S. E. Rep. 479, Supreme Court of Appeals of Virginia. June 16, 1927.

circumstances, can reach no other conclusion than that reached by the commission, *viz.*, that the sarcoma was either caused by the injury or was aggravated by it. . . .

"To this we may add that the courts have in general found no difficulty in cases similar to the one we are considering here, in applying the ordinary rules of evidence, and in drawing the ordinary conclusions of cause and effect from established facts, and we find none. This, we doubt not, courts will continue to do with a full sense of justification and without apology until the cause of cancer is definitely and scientifically established."

In the following case of intra-abdominal sarcoma following a recent trauma the evidence of a causal relationship appears to be convincing.

CASE V.—R. T., male, aged thirty-four years, had always been in good health until July 3, 1916, when he fell from a building for a distance of eighteen feet striking on a cement floor; he landed in such a position that his upper abdomen received a sharp blow from his doubled-up elbow. Six or seven months later he complained of pain in the upper left abdomen at the site of the injury. He consulted a number of physicians and surgeons who made various diagnosis. In December, 1917, he came under the care of Doctor Charles H. Mayo, who made a clinical diagnosis of lymphosarcoma of the small intestine. He performed an exploratory operation which revealed a large, inoperable tumor of the mesentery and small intestine, largely posterior to the parietal peritoneum. Deeming a surgical removal unwise, Doctor Mayo referred the patient to us for conservative treatment. Under irradiation and toxins the tumor practically disappeared, and the disease was held under control for five or six years at the end of which time metastases developed in the neck and axilla. Under further treatment the disease was again controlled. At the end of nine years the patient had a recurrence of the original tumor and died in a few months. The microscopical diagnosis in this case was lymphosarcoma. Autopsy was performed, and the only tumor found in the entire body was at the site of the original tumor.

Trauma and Its Relationship to Tumors of the Testicle.—Most writers on tumors of the testicle, having observed a large number of cases in which there was a history of antecedent local trauma, have come to the conclusion that there is a causal relationship between trauma and tumor formation. Dew,[18] in his book on "Malignant Disease of the Testicle," states:

"The testicle from its exposed situation is particularly prone to traumatic insults, and as it is peculiarly sensitive these are often keenly remembered, yet neoplasms of the testicle are quite rare. Still even bearing in mind the very human tendency which seeks to attribute disease to a definite cause such as injury, all statistics go to show that, in this organ, trauma is an important factor, and most writers on the subject emphasize its importance.

"In a carefully recorded series Howard[19] found that eight cases out of twenty-seven gave a history of recent trauma and in another case there was a history of trauma some time before. Miyata,[20] out of twenty cases, found trauma a factor in ten. Sehaguchi reported four out of thirty-two to give a definite history of injury. O'Crowley[21] reported six cases out of a series of thirteen. In the present series I have found that out of the thirty-three of which clinical notes are available, twelve gave a history of more or less recent trauma.

"Practically all observers give similar figures. . . .

"It is extremely difficult to be sure that a definite essential connexion between tumour formation and trauma does exist, but the figures strongly favour that belief. It is well known that, experimentally, trauma has the power of exciting spontaneous growth in ova

parthenogenetically and it may be that investigation along these lines will provide an explanation.

"There is no doubt that the opinion of experienced observers may be summed up by stating that there is a definite history of trauma in anything up to 50 per cent. of these tumours, though definite causal relationship still remains to be proved."

According to Öphuls, carcinoma of the testicle is the only type of carcinoma which is frequently caused by a single, more or less severe injury. He states: "This includes the so-called round-celled sarcoma of this organ, because careful histological examination reveals that most of these so-called sarcomata arise from the epithelium of the seminiferous tubules, and therefore should be classified as carcinomata. The frequency of their traumatic origin, to my mind, has not been sufficiently emphasized; but anyone who has had experience with these growths will readily confirm it from personal experience, and a study of the case reports collected in literature reveals the same thing. When we consider the constant and very active multiplication of the spermatogenous cells under normal conditions, we may readily understand why a thorough, even single disturbance of them may lead to such disastrous consequences. The mere fact that these growths are usually encountered in comparatively young individuals, in the prime of sexual activity, lends strong support to this theory. . . . It would appear, then, that in estimating the probability of a connection between trauma and the development of a true tumor, the collective experience so far obtained in the particular type of tumor concerned should also be carefully taken into account."

In a monograph on "Malignancy of the Testis, with Special Reference to Undescended Testis" (Minneapolis Surgical Society Prize Winning Essay for 1930), Rea[22] reports seventy-six cases of malignant tumor of the testis. In discussing etiology, Rea[22] states: "Twenty-nine of the patients (38 per cent.) gave a history of some variety of trauma preceding the recognition of the tumor, but the information in the records is of such a character as to leave much doubt as to whether the trauma had actually any significance in the development of the lesion or whether it served merely to call attention to a pre-existing tumor."

Kober[23] found a history of trauma in 28 per cent. of 114 cases.

One of the writers (W. B. C.), in a study of sixty-four cases of sarcoma of the testis personally observed up to 1914, found a definite history of antecedent trauma in 33 per cent.

Melanotic Sarcoma.—While the majority of melanomas or melanotic sarcomas have their origin in a pigmented mole, the transformation of the latter into a malignant tumor is usually associated with repeated trauma or repeated irritation, for example, friction from clothing or from a bath-towel. Many cases, however, give a history of a single local trauma, such as, tying off a pigmented mole with a silk ligature, the use of cautery or some form of caustic. One striking example in our experience of a melanoma developing from a single trauma occurred in an Army man who, in 1917, received a typhoid inoculation through a small pigmented mole in the deltoid region. This had existed since childhood. Within a few weeks the mole showed evidence of increasing activity; it grew rapidly in size, and in spite of a surgical removal, the disease metastasized to the glands proving fatal in less than a year.

Neurogenic Sarcoma.—The statement has been made at medico-legal trials that neurogenic sarcoma is practically never associated with antecedent trauma. This has not been borne out by our personal experience. We have

observed a number of cases of neurogenic sarcoma in which there was a very definite history of local trauma. For example, a woman while travelling on an ocean liner was struck in the middle of the forearm by the heavy wooden cover of a wash bowl. There was a definite bruise but no swelling at the time. A few months later, a swelling developed at the exact site of the injury. A local removal was made, followed later by an intrascapulo-thoracic amputation. The disease metastasized to the lung, proving fatal within a year.

An analysis of seventy-two cases of neurogenic sarcoma by Quick and Cutler[24] shows a history of trauma in fourteen, or 19.3 per cent.

Conclusions.—A careful study of our own series of cases personally observed, we believe, warrants the following conclusions:

(1) That a single local trauma may be an important factor, probably the determining factor, in the development of malignant tumors of all types.

(2) That trauma is a causative factor in a larger proportion of cases of sarcoma than carcinoma, and in a larger proportion of bone sarcomas than soft-part sarcomas.

(3) That the interval of time elapsing between the injury and the appearance of the tumor is often much shorter than is recognized by most writers. In the majority of cases the tumor develops within the first month or six weeks of the injury but in a considerable number of cases it may develop within one or two weeks. The latter cases justify the classification of *acute traumatic malignancy* originally suggested by the English surgeons. The examples herein reported furnish convincing evidence of the actuality of such a condition.

(4) While courts and compensation bureaus both in this country and in Europe have very generally recognized single trauma as a competent producing cause of all types of malignant tumors, it is only fair to the insurance carriers that each case be studied and judged on its own merits.

(5) If the case in question fulfills all the conditions laid down by Segond then a causal relationship between the injury and the tumor must be admitted.

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SURGICAL OPERATIONS IN ADDISON'S DISEASE

SUCCESSFUL EPIDIDYMECTOMY AND ORCHIDECTOMY FOR TUBERCULOSIS

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FROM THE DIVISIONS OF MEDICINE AND SURGERY OF THE MAYO CLINIC

A PATIENT with Addison's disease requires protection from every outside influence which will increase the demand on his slender reserves of strength. Undue fatigue, extremes of heat or cold, and mental or emotional strains are all to be avoided. The increased susceptibility to infection and the frequency with which fatal termination of the disease are initiated by intercurrent infections such as colds, sore throat, or influenza are well recognized. It is not surprising, therefore, as pointed out by Rowntree and Snell,[4] in 1929, that these patients are extremely bad risks from the surgical point of view. We have observed fourteen cases in which surgical procedures have been attempted in patients with Addison's disease. (Table I.) In eleven of these cases, the shock of operation precipitated an attack of acute suprarenal insufficiency with crisis and death from one to sixteen days later. Six of these patients presented themselves to the surgeon because of tuberculosis of the genito-urinary tract. In only one was the possibility of Addison's disease suspected prior to operation but in all extensive and previously unsuspected caseous tuberculosis of both suprarenal glands was found at necropsy. Apparently it is of the greatest clinical importance to consider fully the possibility of suprarenal tuberculosis with latent Addison's disease as a complication among patients who present themselves for the surgical treatment of tuberculosis of the genito-urinary tract.

In the last two years we have had the opportunity to treat thirty-two patients with Addison's disease with the cortical hormone of the suprarenal gland prepared by the method of Swingle and Pfiffner.[5, 6, 7, 8] This experience has convinced us of the efficiency of this preparation in combating crises of acute suprarenal insufficiency and the effects of acute streptococcal sore throat in such cases. Besides the treatment of the general condition, which resulted in improvement, several of these patients have had teeth extracted, one had a minor nasal operation and another a plastic operation on the eyelid while under treatment with the cortical hormone. These minor operations caused no more discomfort or reaction than they would if the patients had been in normal health, a striking contrast to the severe reactions which usually characterize such procedures in untreated cases of Addison's disease. The effect of treatment in these cases justified its trial in a more serious surgical condition.

TABLE I

Results of Operation

Case	Age, Years, and Sex	Asthænia, Grade	Pigmentation, Grade	Blood-pressure, mm. of Mercury		Operation	Comment	Necropsy Data
				Systolic	Diastolic			
1	34, M.	2	1			Amputation of right leg	Death in three days	Bilateral tuberculosis of suprarenal glands
2	46, M.	2	2	108	78	Cholecystectomy	Death in one day	Bilateral tuberculosis of suprarenal glands
3	37, M.	1 to 2	1 to 2	130	90	Thyroidectomy	Death in three days	Atrophy of suprarenal glands
4	40, F.	0	0	140	60	Hysterectomy	Death in three days	Atrophy of suprarenal glands
5	32, M.	0 to 1	1 (racial)	105	75	Hæmorrhoidectomy	Death in four days	Bilateral tuberculosis of suprarenal glands
6	65, M.	1	1	110	78	Nephrectomy	Death in one day	Bilateral tuberculosis of suprarenal glands
7	26, M.	1 to 2	2	105	75	Nephrectomy	Death in three days	Bilateral tuberculosis of suprarenal glands
8	32, F.	3	2	75	?	Nephrectomy	Death in two days	Bilateral tuberculosis of suprarenal glands
9	M.	1	0			Cystostomy	Death in three days	Bilateral tuberculosis of suprarenal glands
10	25, M.	2	1 to 2	100	80	Orchidectomy	Death in seven days	Bilateral tuberculosis of suprarenal glands
11	30, M.	2 to 3	1	106	66	Bilateral epididymectomy	Death in sixteen days	Bilateral tuberculosis of suprarenal glands
12	54, F.	2	3	80	60	Drainage of lumbar abscess	Survived, Addison's disease	
13	52, M.	2	2	100	75	Excision of cervical lymph-nodes	Survived, Addison's disease	
14	54, F.	2	2	90	70	Tonsillectomy	Survived, Addison's disease	

REPORT OF CASE.—A man, aged thirty-two years, came to The Mayo Clinic, April 29, 1931, because of a generalized brownish pigmentation of the skin which had first been noted following an attack of influenza three years before. Apart from the color of the skin he had been well and active, and had not lost weight or strength. Examination showed a diffuse brown pigmentation of the skin with intensification over the scrotum and perineum. There were a few black freckles scattered over the face and neck. The lips were dark, but the buccal mucosa was not pigmented. The blood-pressure in millimetres of mercury varied from 98 to 134 systolic and from 68 to 80 diastolic.

Apart from marked chronic prostatitis, the results of examination were negative. No evidence of tuberculosis was found on general examination, by röntgenograms of the thorax and suprarenal area or by search of the sputum, urine or stools. A diagnosis of probable Addison's disease was made on the basis of the pigmentation. The patient was advised to conserve his strength and was placed on a Muirhead regimen by way of protection and treatment. Treatment for the prostatitis was also ordered.

The patient returned to the clinic January 8, 1932, because of acute left epididymitis which had developed two days before. Apart from the epididymitis his general condition was the same as the year before. No further evidence of active tuberculosis could be found. The acute inflammatory manifestations of the epididymitis at first subsided under conservative treatment, with rest, hot applications, and calcium gluconate, but later recurred. February 1, acute left hydrocele also developed. In consequence of this it was felt that the lesion was probably tuberculous and that operation was indicated. A pre-operative course of treatment with the suprarenal cortical hormone (Eschatin, Parke, Davis and Company) was decided on. The patient was given forty cubic centimetres in divided doses intravenously during the two days preceding operation.

FIG. 1.—Tuberculosis of the epididymis with slight involvement of the testis.

At operation, February 9, which was done under local anæsthesia, it was found that the left testis was enlarged to about twice normal size, due to enlargement and partial destruction of the epididymis by multiple tuberculous abscesses. The inflammation appeared to extend into the testis and rather than take the risk of failing to remove all of the areas of infection as well as to avoid the effects of absorption from an infected area in the testis, it seemed safer to remove the testis with the epididymis. This was done through a scrotal incision. The pathologist reported tuberculosis of the epididymis with slight involvement of the testis. (Figs. 1, 2 and 3.)

Immediately after the operation, the patient was given twenty cubic centimetres of the

FIG. 2.

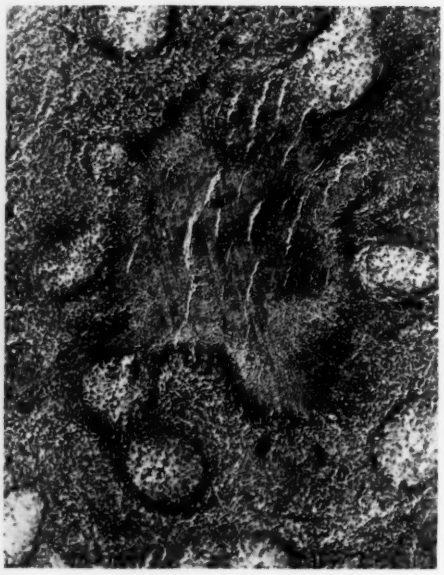

FIG. 3.

FIG. 2.—Tuberculosis of the epididymis with caseation (x 60).
FIG. 3.—Tuberculosis of the testis with caseation (x 60).

cortical hormone. He had a comfortable night and the next day two injections of ten cubic centimetres each were given. Subsequent injections were given at longer intervals, the total amount being 110 cubic centimetres. As shown in Fig. 4, the post-operative

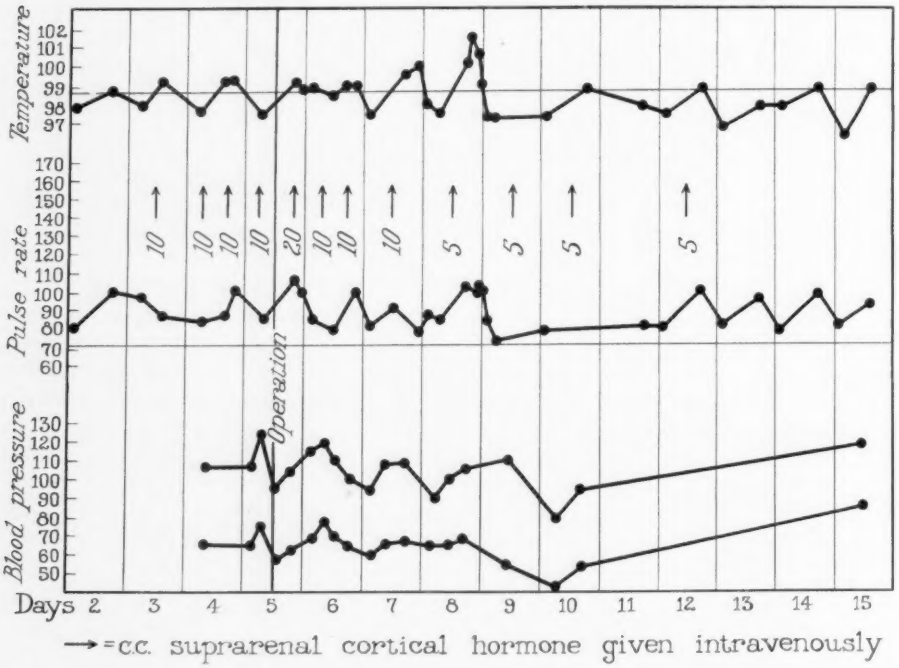

FIG. 4.—Temperature, pulse rate, and blood-pressure readings before and after operation. Arrows indicate intravenous injections of the suprarenal cortical hormone.

course was entirely uneventful. The blood-pressure never fell below 90 systolic, varying between that and 125 with a pressure of 50 to 78 diastolic. He was dismissed from the hospital February 24 in good condition and is now back at work.*

When the patient was first seen, a diagnosis of probable Addison's disease was made. Observation over a period of nine months together with the demonstration at operation of active tuberculosis of the genito-urinary tract, we believe, has now established the diagnosis of Addison's disease beyond any reasonable doubt.

We have been unable to find in the literature record of a successful major operation on a patient with unquestioned Addison's disease. Oestreich,[3] Bartels,[1] and Heinlein[2] each have reported the removal at operation of a single caseous suprarenal gland, but in none of these cases was their clinical signs of suprarenal insufficiency and the presumption that the destruction of the suprarenal gland was primarily unilateral is likely.

This case is unique in that, so far as we know, it is the first one in which pre-operative and post-operative treatments with the cortical hormone have been used to protect the patient against the shock of operation, and the only case of Addison's disease, to our knowledge, in which the patient survived such an operation.

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* The patient returned to the clinic September 15, 1932, for reëxamination. He was in excellent condition, had gained in weight and the pigmentation of his skin did not appear as marked as on his previous visit.

SPONTANEOUS PNEUMOTHORAX*

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Case I.—A man, aged twenty-five years, by occupation a law clerk, was admitted January 8, 1930, to the Misericordia Hospital in the service of Dr. George P. Muller, with the diagnosis of acute mesenteric ileus or chronic appendicitis. His chief complaint was severe upper abdominal pain. The patient was awakened in the early morning with severe sharp stabbing pain in the epigastrium which was not referred and which doubled him up. He had normal bowel movement, but obtained no relief from pain. Shortly afterwards, he had a copious watery bowel movement, but still with no relief from pain. The pain became worse and he called a physician, who gave him morphine hypodermically, but without relief. He was nauseated but did not vomit. The family physician stated that the abdomen was rigid and tender and from this clinical picture he made a diagnosis of ruptured peptic ulcer and sent him immediately to the hospital.

Physical Examination.—Blood-pressure, 130 S, 75 D. Temperature, 96.3; pulse, 126; and respirations, 40. The patient was suffering agonizing pain, his skin was cold and clammy, but he had no dyspnœa or cyanosis. On examination of the chest, expansion was free and equal; no respiratory embarrassment; tactile fremitus normal; percussion note resonant throughout and breath sounds vesicular. Heart: No murmurs or arrhythmia. Sounds of good quality. Pulse: Rapid, of good volume and rhythm.

Abdominal examination was negative, except for board-like rigidity and extreme tenderness in epigastrium.

Pre-operative diagnosis: Ruptured gastric ulcer.

Immediate operation was performed under spinal anæsthesia. Spinocaine, four cubic centimetres, was given, but the abdomen still showed board-like rigidity although he did not feel any pain. Anæsthesia was re-enforced with ether.

On opening the peritoneum no free fluid was encountered. The stomach was found greatly dilated, but revealed no evidence of ulcer or of perforation. The entire abdominal cavity was explored, but no cause for his symptoms was discovered. A gastrotomy was performed to empty the dilated stomach of gas. The gastric opening was then closed. The cæcum was located and delivered and a chronically diseased appendix removed. The abdominal wound was closed without drainage. The patient left the operating room in good condition.

Post-Operative Convalescence.—Twelve hours after operation, the patient became dyspnœic with shallow respirations. He was cyanotic, felt that he was suffocating, and complained of pain in his left scapula. He was cold, clammy, restless, and at times irrational. Examination of chest showed that the left side was fixed, the intercostal spaces were slightly bulging, the percussion note was tympanic and the heart was pushed to the right. The abdomen was soft but not distended. A diagnosis of left spontaneous pneumothorax was made. An X-ray was taken immediately which confirmed the diagnosis.

The patient was desperately ill and immediate aspiration of the chest was instituted. The cyanosis became less, the pulse of better quality and the rate slower.

Before Aspiration	After Aspiration	24 Hours Later
Pulse Rate 140	120	90
Temperature ..... 101° F.		99° F.
Respirations 46	40	28

* Read before the Philadelphia Academy of Surgery, December 5, 1932.

The patient made an uneventful recovery, although at the time of discharge he still showed signs of pneumothorax with some displacement of the heart.

Progress Notes Since Discharge from Hospital.—After the patient's discharge from the hospital, he felt well; had no dyspnœa, and X-ray examination of chest for comparative study with previous films showed the pneumothorax with collapse on the left side to have entirely disappeared. The entire right pulmonary field showed a slight mottling throughout. This had not the characteristic appearance of tuberculous infection, but rather that most often produced by an influenzal infection, or secondary to upper respiratory disease. About two weeks later, he complained of some dyspnœa and tired feeling, and was admitted to the hospital of the University of Pennsylvania for study on March 3, where it was stated that a recurrence of the pneumothorax had occurred.

Since then there has been no recurrence of the pneumothorax.

The speaker remarked that spontaneous pneumothorax of the complete type is rarely seen in an individual who does not show any evidence of intrathoracic disease. It was best defined by Hammon as a "pneumothorax coming on in an apparently healthy individual without any ascribable cause, resulting in no infection of the pleura and therefore unaccompanied by constitutional symptoms and healing rapidly and completely in a few weeks." The existence of it has been known for years. Hippocrates called it empyema, and it was again brought to medical attention by Meckel in 1759; in 1803, Ward gave it a definite name; Laennec in 1819 described a case; and Zahn in 1891 published the first full satisfactory report.

The onset of pneumothorax may be insidious, slow leak, or acute, the acute causing respiratory embarrassment. The description of the physical and clinical signs can be found in any text-book of medicine. Kahn[6] states that a small pneumothorax containing only 200 to 500 cubic centimetres is practically undetectable by physical examination, and one of 200 to 250 cubic centimetres even by Röntgen examination. This he has verified by examination after initial introduction of air in artificial pneumothorax therapy.

Pneumothorax is further divided into tuberculous and non-tuberculous or idiopathic types, for which no set cause can be found.

Weber[5] gives a review of causes of idiopathic, believing some due to rupture of emphysematous bleb in the margin of the lung, and some due to inflammatory condition causing adhesions between the visceral and parietal pleura, and that some unusual exertion causes adhesions to be torn loose, thus causing a rupture, the exciting causes being coughing, sneezing, laughing, vomiting, hiccough, muscular strain. Further, some may occur during sleep. Wood and Vinson[4] report one due to forced deglutition occurring in a case of obstruction of the cardiac end of the stomach.

Other causes, but not idiopathic, are abscess; Browder[14] reports a case following rupture of subpleural abscess; gangrene and new growths of lung; bronchiectasis, emphysema, tuberculosis, empyæma and pneumonia, accidental pneumothorax following fracture of ribs. Harvey[16] reports a case.

Weber[5] believes that all cases are due to healed miliary tubercle, rupture

of minute superficial emphysematous bulla, just below the visceral pleura; that the difference between idiopathic and tuberculous spontaneous pneumothorax is that the latter are connected with still more or less virulent tuberculous foci containing living organisms, whereas the latter are connected with healed non-virulent sterile lesions.

Heischboeck[11] does not believe that tuberculosis always causes the small bleb, but that other lesions can cause it. Burrell[8] noted in his work that in spontaneous idiopathic pneumothorax pleural effusion does not occur, and in a large series of cases in which artificial pneumothorax is produced that effusion occurs in tuberculosis cases and not in the non-tuberculous cases and that this can be used as a diagnostic point. Kahn[6] gives the types of pneumothorax with treatment and divided thus:

(1) *Closed type*, in which there is little or no respiratory distress. These cases do best if left alone, and the air will be absorbed.

(2) *Closed pneumothorax* with symptoms of respiratory distress. In these, aspiration of air should be carried out.

(3) *Closed pneumothorax* with effusion. The treatment is expectant. Aspirated for study of the fluid and air drainage instituted if necessary.

(4) *Valvular pneumothorax.*—These are, as a rule, fatal. The treatment of this type of case is by continuous air drainage through a needle with rubber tube passing to some fluid container (closed drainage). Pyothorax usually occurs.

(5) *Open pneumothorax.*—In these cases, as pyothorax occurs, postural drainage should be instituted.

In review of cases by Heischboeck, he noted that the occurrence of spontaneous pneumothorax occurred between the ages of fifteen and forty-five. Nikolsky stated that in 80 per cent. of cases, males are affected, and it occurs with equal frequency on both sides of the chest, though individual statistics vary on this point. Palmer and Taft[2] showed that tuberculosis in adults caused 80 to 90 per cent. of the cases in adults, and 40 to 50 per cent. in children.

The duration of the pneumothorax in the uncomplicated cases is, as a rule, from two weeks to two months; however, it can last longer. Heischboeck reports a case of eleven years' duration; Wieler, one of twenty years; LeWald,[13] one of ten years, and another of one year.

Spontaneous pneumothorax can recur. Watson and Robertson[12] report three cases.

Biach reported 918 cases of spontaneous pneumothorax, but 715 were proved to be tuberculous, and 200 cases were idiopathic. Fussel and Reisman reported fifty-eight cases with one death. Biesenthal and Snyder,[3] in 1932, reported 200 cases on record and added twelve cases of their own.

In going over the literature, spontaneous pneumothorax of idiopathic division is not uncommon.

It was interesting to note that Heischboeck reports fifteen cases of bilateral pneumothorax with recovery; however, many cases of bilateral pneumothorax nearly always prove fatal, the patient dying in a few hours.

In reference to treatment: That of Kahn has already been given. Stewart[1] advised tapping of chest and leaving needle *in situ*. In one case, he let the needle remain for three weeks. Biesenthal and Snyder advise rest in bed for a period from four to six weeks. By the end of that time, the lung will re-expand. In one of his cases, re-expansion of lung did not occur after four months, and he hastened the expansion of lung by use of blowing in bottles. Weber believes that all get well without specific treatment. Tilton and Schroeder[7] advise immediate aspiration in cases showing acute symptoms. Burrell[8] states in cases of few symptoms, the lung usually re-expands in a few days and a period of rest is all that is required. If the symptoms persist remove gas in order to assist re-expansion and stop if patient coughs. If pressure is lowered too quickly, the patient develops paroxysms of cough with frothy expectoration and very severe dyspnœa lasting several days.

Fogelberg,[9] in 1924, had two cases in which he hastened re-expansion by Spengler's method of interpleural injection of 30 per cent. solution of glucose. This method is to be used only in spontaneous pneumothorax without effusion.

The following is a case report of acute spontaneous pneumothorax which simulated acute upper abdominal lesion. This case is reported not so much because of the rarity of the occurrence of cases of this kind, but because of the error in diagnosis which led to a major operation.

This case is interesting because it seems probable that the first rupture with escape of air filling the mediastinal tissues and through contact with the nerve supply gave the intense symptoms of referred upper abdominal pain. Later, the rupture into the pleural cavity occurred with the development of pneumothorax. It is also interesting to note that at the time of the first pneumothorax the patient's symptoms were intense and particularly he had the fear of impending death. Two and one-half months later, when he had his second attack, he was conscious only of pressure, but otherwise was not disturbed. Since that time, two and one-half years, he has not had a recurrence.

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LATERAL ABERRANT THYROID GLANDS

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Shrager,[1] in 1906, defined an aberrant thyroid as a mass of tissue having the structure of a normal or pathological thyroid gland, and situated at some definite distance from the normal thyroid, with which it has no connection. He suggested that accessory, allied, and other synonyms for these entities are not desirable terms, but that the most appropriate is "aberrant thyroids," indicating segments which are separated from the parent thyroid.

Fifty-one cases of lateral aberrant thyroid glands have been reported to date. J. V. Leech, L. W. Smith, and H. M. Clute[2] maintain that the paucity of reported cases is due to a failure of many operations to reach the literature, or to a failure to recognize the true nature of these cervical tumors. The diagnosis is usually made by the pathologist. These lateral aberrant thyroids are usually the seat of pathological lesions such as neoplastic formations or inflammatory changes. Billings and Paul[3] report that 70 per cent. of lateral aberrant thyroid tumors are of the papillary type. The case here reported is one of an intracystic papilloma of a lateral aberrant thyroid gland.

Case I.—H. S., male, aged forty-one years, first consulted us September 30, 1931, complaining of a lump on the left side of the neck of eleven weeks' duration. There was no history of pain, loss of weight, or of a tendency toward progressive enlargement of the tumor. Dysphagia and other pressure symptoms were never noted. The past and present history was irrelevant. Examination revealed a mass about the size of an apricot situated on the left side of the neck over the upper portion of the sternocleidomastoid muscle, and extending up to the lobule of the ear. This mass was firm but elastic, and although freely movable from side to side was apparently non-mobile from above downwards. The skin was not attached to the tumor at any point. Tenderness could not be elicited, nor could a communication with the mouth be established. The superficial lymph-nodes and thyroid were uninvolved. Blood smears and white blood-cell count were normal. Blood-pressure was 220 systolic and 110 diastolic. The urine contained a heavy trace of albumin with a few hyaline casts. There was a moderate degree of left ventricular preponderance.

Operation.—(J. A. L.) Under avertin anæsthesia, a three-inch oblique incision was made directly over the body of the sternocleidomastoid muscle. Situated in the upper anterior triangle of the neck and firmly attached to the carotid sheath beneath, and to the external jugular and facial veins above and anteriorly, there was a tumor about the size of an apricot which appeared to have a duct at its upper end terminating in the region of the pharynx. The tumor was cystic with a markedly thickened wall and the contents consisted of a yellowish mucoid fluid containing a number of small golden-brown granules. There were many dense adhesions binding the tumor to the structures comprising its bed.

To free the tumor it was necessary to split the fibres of the sternocleidomastoid muscle. With great difficulty the lower pole of the growth was mobilized and the process

of separation was carried out from below upwards. In doing this, some of the cyst contents escaped into the wound. The entire tumor was extirpated and its pedicle, situated at the upper pole, was divided between ligatures. The wound was then flooded with 50 per cent. alcohol and closed in layers over a rubber dam. The patient made an uneventful recovery and was discharged from the hospital eight days after operation. He was last seen November 11, 1931, and appeared perfectly well, there being no evidence of recurrence.

Pathological report.—The specimen consisted of a cystic mass measuring 3 centimetres in diameter. When opened, its lining presented a number of small papillary projections, and its contents consisted of a thick hæmorrhagic grumous material. On section, hæmorrhagic fibrous tissue was seen with many cystic areas showing papillary projections. The cells showed no cavernous changes. (Figs. 1, 2 and 3.)

Diagnosis.—Papillary cystadenoma of an aberrant thyroid gland.

In order to obtain a clear understanding regarding the mode of origin of an aberrant thyroid, it is necessary to refer back to the embryology of the normal thyroid gland.

The thyroid gland is formed from an evagination of the anterior pharyngeal wall at the level of the second branchial arch. It was originally thought that the thyroid gland had a double anlage, one arising laterally, the other in the mid-line; but the works of Muller, His, Verdun and Tourneaux[4] seem to prove that the thyroid originates from a single median evagination. The thyroid appears in embryos of 4 millimetres as a ventral outgrowth of epithelium in the floor of the pharynx immediately behind the tuberculum impar. This evagination grows into the mesodermal tissue in the ventral wall of the neck, forming a transverse mass of epithelium. This mass breaks into irregular cords of cells which by a further process of budding grow caudally along the anterior surface of the larynx. This mass of tissue is solid and may assume the appearance of a tube but never really becomes one, although it is designated as the thyroglossal duct. This solid mass grows caudally and divides into a right and left lobe which are united by a strand of tissue identical in structure to the lobes which cross over the trachea. The thyroglossal duct may retain its connection with the isthmus for variable periods, reaching usually to the foramen cæcum at the root of the tongue. This duct is customarily divided into a lingual portion which extends from the foramen cæcum to the hyoid bone, a hyoid portion lying in the body of the hyoid bone, and a thyroid portion extending from the hyoid bone to the isthmus of the thyroid gland.

Concomitant with the full development of the thyroid, the thyroglossal atrophies into a fibrous cord which is often designated as the tractus thyroglossus. Occasionally, the duct fails to become fully obliterated, resulting in the formation of thyroglossal cysts. Cases of failure of closure of the thyroglossal duct are frequently associated with the presence of rests of thyroid tissue situated along the course of the duct. Occasionally, the duct may terminate in the anterior mediastinal space, in which event aberrant nodes may be found in that location.

The exact origin of lateral aberrant thyroids is still a mooted question.

FIG. 1.—Portion of tumor showing proliferation of the germinal centres. (Low power.)

FIG. 2.—Many cystic areas with papillary projections of embryonic origin. (High power.)

FIG. 3.—Portion of tumor showing proliferation of the germinal centres. (High power.)

Some observers are of the opinion that fragments of the thyroid may become detached during the gland's descent from the neck. The best explanation is that given by Grosser,[5] who maintains that these structures develop from clusters of cells arising from the posterior aspect of the fifth branchial pouch. (See Fig. 4.) These cell clusters, in their migration, may fail to meet and fuse with the thyroid proper, and later, as shown by Virchow, they become activated and give rise to tumors.

Seventy per cent. of these aberrant thyroids give rise to neoplasms of the

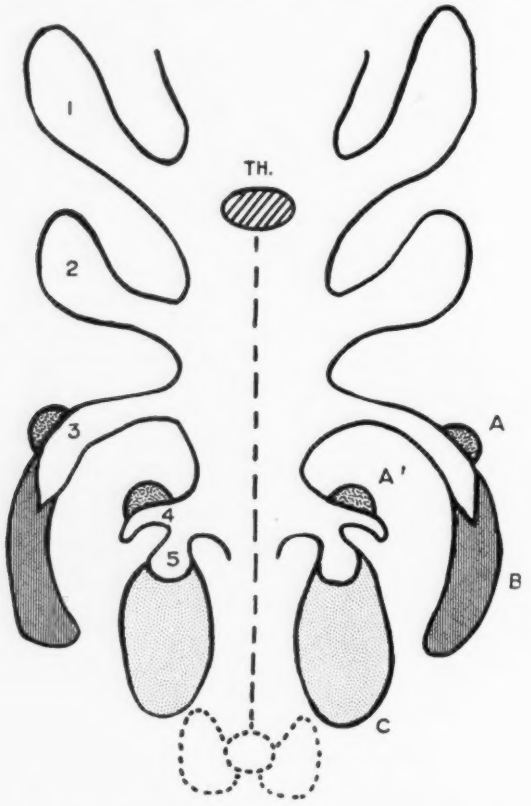

FIG. 4.—1—First branchial groove; 2—second branchial groove; 3—third branchial groove; 4—fourth branchial groove; 5—fifth branchial groove; A—thymic portion of the parathyroid; A′—thyroid portion of the parathyroid; B—thymus; C—post-branchial body; TH—thyroid. (From Crotti's Thyroid and Thymus. Lea and Febiger.)

papillary type. The other lesions are papillary adeno-carcinoma, epithelioma, alveolar carcinoma and carcinoma. The tumor is usually slow-growing and subject to involutional changes such as cystic degeneration, hæmorrhage and calcification. A well-defined capsule is usually present. They may undergo malignant changes and give rise to metastases. Although the vast majority of these tumors are found in the neck, they may occur in the bones, pleura, pericardium and ovaries. J. B. Murphy[6] believed that aberrant thyroid nodules were metastatic in character. This view, however, has not been uni-

versally accepted because of the failure to find malignant changes in the thyroid proper. Aberrant thyroids may be found anywhere in the lateral regions of the neck. The most common location is in the mid-portion of the neck beneath the sternocleidomastoid muscle. In a number of cases they are situated in close proximity to the angle of the jaw, and in others may be found lying in the supraclavicular fossa. In Billings and Paul's series of collected cases the tumors occurred in 75 to 80 per cent. of the cases in females. The average age was thirty-four for females and forty-two for males. The youngest case was in a girl of twelve years, and the oldest in a woman of sixty years.

Affection occurring within the thyroid frequently produces similar changes within the aberrant rests; and conversely when adenoma is suspected within an aberrant thyroid, the thyroid proper should be carefully inspected for a similar disease.

Differential diagnoses.—One must differentiate lateral aberrant thyroids from: (1) Carotid body-tumors; (2) tuberculous glands; (3) branchial cysts; (4) Hodgkin's disease; (5) secondary or metastatic carcinoma; (6) lymphosarcoma; (7) lymphatic leukemia; (8) syphilitic glands.

Tumors of the carotid glands are of infrequent occurrence. The carotid bodies are situated at the bifurcation of the common carotid artery. Microscopically, they are classified as endothelioma or perithelioma, and show low-grade malignant properties early in their development, but later tend to become actively malignant. Their growth shows a slow and progressive course. Tuberculous nodes occur usually in younger patients and are as a rule bilateral. They are multiple and have a tendency to become adherent to one another and to the skin overlying them. Diagnosis can be established by surgical measures, such as removing a node for biopsy. When spontaneous necrosis occurs, leading to a discharging fistula, the diagnosis of tuberculous nodes is self-evident. Confirmatory evidence of an old tuberculous lesion in joints, spine and peritoneum may be elicited. Absence of pulmonary signs is not at all unusual in these cases. Lateral embryonic remnant cysts are due to imperfect closure of branchial clefts and are sometimes known as branchial cleft cysts. The contents of these cysts may be serous, mucous, gelatinous or may resemble the contents of a sebaceous cyst, sometimes containing cell detritus, epithelium and frequently cholesterin crystals. The lining membrane of these lateral cysts as well as aberrant thyroid cysts is epithelial.

CASE II.—(*Branchial Cyst.*) A. W., female, aged twenty-nine years, first consulted us February 22, 1932, complaining of a lump on the right side of the neck. A pre-operative diagnosis of a possible lateral aberrant thyroid was made but the diagnosis of branchial cyst was later established by microscopy. The tumor, of three months duration, had grown progressively larger until it had reached the size of a small hen's egg. There were no pain, no dysphagia, no gastro-intestinal symptoms and no loss of weight. One week prior to the consultation the patient was receiving Röntgen therapy which caused a marked increase in size of the mass. The family history was negative. As to her past personal history, there was nothing of importance except an occasional attack of

cervical adenitis years before. She was well nourished, not appearing acutely ill. Eyes, ears and nose showed no disease. The mouth revealed large and cryptic tonsils. The larynx appeared normal. The heart and lungs were negative. The blood-pressure was 130 systolic and 80 diastolic. A röntgenological examination of the chest showed a few increased hilus shadows and some calcified nodes.

There was a mass about the size of a large English walnut on the right side of the neck in front of the upper portion of the sternocleidomastoid muscle. The mass was hard and gave a sense of slight fluctuation and could be moved upon the deeper structures. It was not attached to the overlying skin, and there was no tenderness elicited. The trachea was pushed to the left of the mass. The other glands and the thyroid were not involved.

The hæmoglobin was 70 per cent. (Sahli); red blood-cells, 4,700,000; white blood-cells, 12,000; and the differential blood smear was normal.

Operation.—(J. A. L.) The tumor was removed February 23, 1932. A three-inch vertical incision along the anterior margin of the sternocleidomastoid muscle was made. Situated beneath the sternocleidomastoid muscle and lying upon the large vessels of the neck in the anterior triangle and extending up toward the mastoid process there was a tumor about the size of a small hen's egg. The tumor was intimately adherent to the muscle and the carotid sheath. The tumor was mobilized from its bed and removed with great difficulty. In its removal, the tumor was opened and a considerable purulent discharge spilled into the wound. The wound was then bathed with weak iodine and alcohol, and a small tube was introduced for drainage. The muscles were closed with interrupted plain catgut sutures, the fascia with interrupted chromic, and the skin with interrupted silk. The anæsthesia administered was avertin, which was supplemented by gas and oxygen. The patient made an uneventful recovery and was discharged from the hospital one week after the operation.

Pathological report.—The specimen consists of a cystic tumor mass that measures 4 by 2½ centimetres. On section the mass is filled with whitish mucoid material and the wall is thick and gelatinous in character, with a few small papillomatous projections toward the lumen.

Histological section of the cystic tumor shows the cyst-wall to consist of squamous epithelium, possibly embryonic in origin, with much lymphoid tissue congestion and some necrotic areas. Many pus cells are seen. There are no giant cells or other signs of tuberculosis. *Diagnosis.*—Branchiogenic cyst (infected).

So closely did this case simulate the first one that it was with exceedingly great difficulty that the differential diagnosis was made, even on histological section.

Hodgkin's nodes are usually multiple, discrete and fail to undergo softening as in the case of tuberculous nodes. There is also a failure of the nodes to coalesce or to become adherent to the skin or deeper parts, or to form a fistulous tract. Splenic enlargement is frequently associated with Hodgkin's disease. Diagnosis is made by histological study of the extirpated node.

Secondary or metastatic nodes in the neck frequently simulate aberrant thyroids, and when found, one must always think of a primary lesion in the neighborhood. This lesion is usually a squamous carcinoma of the buccal cavity, lip, tongue, palate or the pharynx, larynx or œsophagus. Carcinoma is a disease of the middle and later periods of life. The rapid enlargement of nodes, their stony hardness, the manner in which they become fixed to the deeper structures and skin leave little doubt as to the diagnosis.

Lymphosarcoma in the cervical nodes is of no infrequent occurrence. In lymphosarcoma, the systemic character of leukemia is missing, and the spleen, liver and marrow are rarely involved. The disease is usually found among robust males between twenty-five and fifty-five years of age. Histologically, the tumor is composed of lymphoid cells of variable size lying in an atypical reticular tissue. Lymphosarcoma can be distinguished from other forms of lymphoma by its local destructive tendency and by the formation of true metastases in distant organs. Leucocytosis is usually present and may be so marked as to suggest leukemia. The excess cells are usually of polymorphonuclear type. X-ray therapy after extirpation of the tumor has often temporarily arrested the progress of the growth.

In lymphatic leukemia there is an increase of lymphoblastic cells accompanied by generalized node enlargement. None of the other conditions exhibits pathognomonic blood changes seen in lymphatic leukemia.

Syphilitic nodes rarely reach great size. They are almond-shaped and firm, painless and do not become adherent to the skin or deeper parts. A history of chancre with a positive Wassermann reaction differentiates syphilis from the other conditions.

The prognosis is good in cases of complete extirpation. Failure to remove the growth may result in malignant changes occurring in it as was seen in the case reported by Wohl,[7] in 1917. In performing the operation, one should aim at the complete extirpation of the aberrant tissue, since failure of removal often results in recurrence. Operative procedures are difficult because of the close proximity of the tumor to important vascular and nerve trunks. The capsule should be included in the extirpation. Following operation, X-ray treatment should be given in all cases.

Summary and Conclusions.—(1) Lateral aberrant thyroids are of rare occurrence. (2) These tumors arise from the fifth branchial pouch. (3) Malignant changes frequently occur within these aberrant thyroids. (4) The majority of lateral aberrant thyroids occur in females. (5) Clinically, the differentiation between branchial cyst rests and lateral aberrant thyroids is impossible. (6) Complete extirpation followed by Röntgen therapy is the procedure of choice in the management of such tumors.

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PROLAPSE OF THE RECTUM

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Considerable divergence of opinion exists regarding pathological changes, classification, and treatment of rectal prolapse; in other words, an external prolapse which one observer considers caused by intussusception of the rectosigmoid, another may describe as true prolapse of the rectum. Prolapse which one observer may classify as grade 3, another may classify as grade 1. Similarly, one surgeon may recommend local or non-surgical treatment, and another may suggest some type of intra-abdominal operation. In an attempt to correlate and classify the basic pathological physiology and various methods of treatment of this condition we have reviewed the cases of prolapse of the rectum observed at The Mayo Clinic in recent years. The cases have been classified as: (1) Prolapse of the rectal mucous membrane; (2) prolapse of the rectum proper (procidentia), which may be graded 1 to 4, depending on the extent of the protrusion; and (3) intussusception of the rectosigmoid.

Prolapse of the Rectal Mucous Membrane.—When prolapse of the rectal mucous membrane occurs, the small ring of mucosa which normally protrudes from the anus during the act of defecation fails to retract on completion of this act. Straining or frequent stools accentuate the protrusion. The mucosa becomes redundant and the submucosa is stretched and relaxed. The position of the other layers of the rectal wall remains unaltered. This condition does not constitute true prolapse of the rectum. Anatomically, it bears a marked similarity to the protrusion of redundant gastric mucosa through the pyloric sphincter into the duodenum. It is most common in childhood or old age.

Occurrence During Childhood.—Prolapse of the mucous membrane is more common during the early years of life than at any other time. Lockhart Mummery[3] reported fifty cases in which the average age was two and a half years; the youngest patient was aged three months and the oldest five years. The prolapse is most prone to develop among poorly nourished children, particularly following debilitating diseases during which a large amount of the ischiorectal fat disappears. Constipation and diarrhœa predispose to the development of prolapse; in the former excessive straining and, in the latter, repeated stools are the contributing factors. Intestinal parasites, whooping cough, rectal polyps and measles are the more commonly associated

systemic conditions. Diarrhœa of any origin is the commonest single etiological factor.

Anatomically, numerous hypotheses have been advanced to explain the development of the condition among children: (1) The weak fixation of the rectal tissues in childhood, the fibrous tissues being more elastic and less firmly anchored than in adults; (2) absence of the sacral curve, the course of the rectum is straighter, in comparison with that of the adult, and (3) the high position of the bladder and uterus of the infant. The true etiological significance of these factors is a matter of conjecture and difficult to determine for any individual case. Only rarely is any developmental defect observed which is directly responsible for the occurrence of this type of prolapse.

Occurrence During Adult Life.—Prolapse of the mucous membrane of the rectum occurring among adult patients is usually associated with some preëxisting pathological condition of the rectum which is at least indirectly responsible for the development of the prolapse. It is usually secondary to some type of disturbance which causes excessive straining at stool. The primary condition may be the result of local or systemic disease. Locally, hemorrhoids, polyps, stricture, atrophic or injured sphincter, are the most common preëxisting conditions. Occasionally, disease of adjacent organs may constitute a predisposing factor. Less often, some type of urinary obstruction may be responsible. Usually the development of prolapse is gradual. The rectal mucosa fails to retract following defecation and the protrusion grows slowly, enlarging somewhat with each act of straining, whether it is sneezing, coughing, voiding or defecating.

Diagnosis.—The condition is usually obvious on inspection of the involved area. A rosette of mucosa is present around the anal margin. The color of the protruded tissue depends on its blood supply, ranging from pink to red, or from purple to black in case of strangulation. The latter complication is very uncommon in this type of prolapse. If the prolapse is recent the tissue is pink and moist; later it tends to become redder and to bleed easily on slight trauma. Occasionally, in a long-standing case, the tissues appear pale and leathery. Distinction from intussusception presenting at the anus is established by inserting a finger in the rectum immediately lateral to the protrusion. This is impossible in mucosal prolapse. Hemorrhoids may be excluded by the absence of a central depression. Polyps most always have a pedicle. In a true prolapse of the rectum proper the entire wall of the rectum descends and the condition may be readily recognized.

Treatment.—The treatment may be medical or surgical. Frequently, in the early case, non-operative measures suffice to effect cure, provided any known etiological factors are eliminated. The essential factors of medical treatment are: (1) Eradication of local causes, dilatation of strictures, treatment of hemorrhoids or polyps, and so forth; (2) establishment of normal bowel habits, with elimination of straining at stool, and (3) general hygienic care and treatment of any coëxisting debilitating disease.

Aside from removal of local causes, the readjustment and regulation of the bowels are of utmost importance. This frequently can be accomplished by attention to the diet and the use of appropriate lubricating oils or laxatives. Violent purgatives should not be employed. Locally, the prolapse should be reduced following each defecation; this is accomplished, following suitable cleansing of the parts with warm water and lubrication with olive oil or liquid petrolatum, by gently compressing and pushing the mucosa upward within the anus. Reduction is maintained between stools by strapping the buttocks firmly together with adhesive tape. Occasionally, when the prolapse has existed for a considerable time, and if there has been some degree of constriction to the blood supply, reduction may be very painful and local anæsthesia may be necessary, although it should be avoided if possible. Excessive downward pressure during evacuation can be partially eliminated by the horizontal position on the back or side during defecation. Massage, mechanical devices, electricity or astringent solutions or ointments are all equally ineffective and are not recommended.

Frequently, all medical measures fail and some type of surgical procedure becomes necessary. Fraser has recently reported fifty cases in which treatment by local injections of alcohol was successful. This method is easy of application and has much to recommend it when productive of so efficient results. One injection is usually sufficient; rarely must a second injection be given. In the technic described by Fraser, the index finger is placed in the rectum to serve as a guide for the insertion of a fine needle which is passed through the mucocutaneous juncture into the region of the submucosa. Approximately 0.2 cubic centimetres of absolute alcohol is injected at four equidistant points around the anus, making a total injection of one cubic centimetre. Following this procedure the buttocks are strapped for twenty-four hours, at the end of which time the bowels are opened. If the injection method is not employed, cauterization may be carried out either with fuming nitric acid or, preferably, with the actual cautery. Excision is one of the surest methods for effective cure, provided the etiological factors are eliminated. This is readily accomplished and is not more disabling than other surgical procedures.

True Prolapse of the Rectum (Procidentia).—True prolapse of the rectum is characterized by the presence of all layers of the rectal wall in the protrusion. Two distinct types or grades may be recognized. One type starts below the reflection of peritoneum on the rectum and the other originates above this level. The former is characterized by absence of a marginal sulcus. (Fig 1.) The prolapse begins at the anal margin and is continuous laterally with the mucocutaneous border. The second type, which begins above the reflection of the pelvic peritoneum, has a definite sulcus laterally, since the lowermost portion (anal margin) of the rectum does not participate in the descent. It is this variety of prolapse which is most representative of true rectal prolapse. In comparison with prolapse of the mucosa only, this type is less common and occurs almost always among adults.

Etiology.—The basic anatomical defects responsible for the development of complete prolapse are more complex than for the incomplete form. The same underlying factors are existing in both types, however, varying only in degree. Considerable anatomical investigation has afforded evidence that complete prolapse of the rectum is similar to hernia. Moschcowitz[4] emphasized the importance of the transversalis fascia and mentioned the ubiquitous presence of this layer of fascia which is always found external to the peritoneum. It lies beneath the large vessels and viscera in the abdomen, and is penetrated only where blood-vessels or viscera normally make their

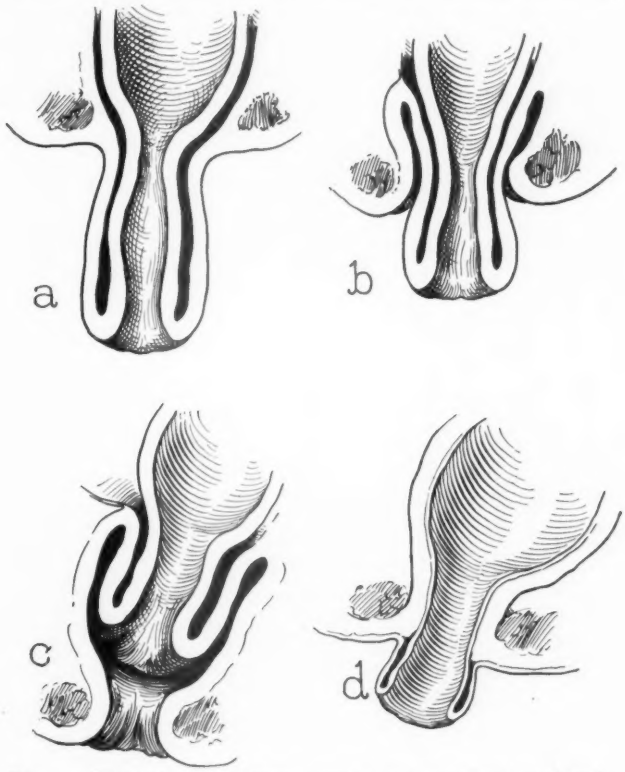

FIG. 1.—Illustration of different grades and types of procidentia.

exit from the peritoneal cavity. As a rule, it is sufficiently strong to prevent herniation. Pathological influences, such as increased intra-abdominal pressure or a pushing force from within, are usually essential to the production of a weakness sufficient to cause herniation. In the case of the rectum, straining as in obstinate constipation, incessant diarrhœa, increased intra-abdominal pressure, trauma incident to parturition, and so forth, are the usual factors responsible for prolapse.

Prolapse of the rectum is similar to a sliding type of hernia. The close adherence anteriorly of the peritoneum to the rectum is the reason, as stated by Moschcowitz, why no true sac exists. (Figs. 2 and 3.) The levator ani muscles and their associated fascia combine with the other parts of the

perineal body to retard the downward protrusion. The musculature of the rectal wall also tends to prevent downward progress of the bowel. However, this is relatively weak and soon yields to the constantly increased intra-abdominal pressure. As the prolapse increases, the posterior wall of the rectum and, later, the sacrum and coccyx prevent its backward extension. The course of the descent is thereby changed and extends at first downward and forward, and finally backward toward the anus until the protrusion appears externally.

Symptoms.—The diagnosis of rectal prolapse usually affords little difficulty. The cardinal features are a protruding mass from the anus (Fig. 4), associated with obstipation and, subsequently, in the advanced cases, with incontinence. The protrusion may vary in size from a few inches to one of considerable length. In the early cases, manual reduction is possible, but in the later cases it is followed by immediate recurrence. At first the pro-

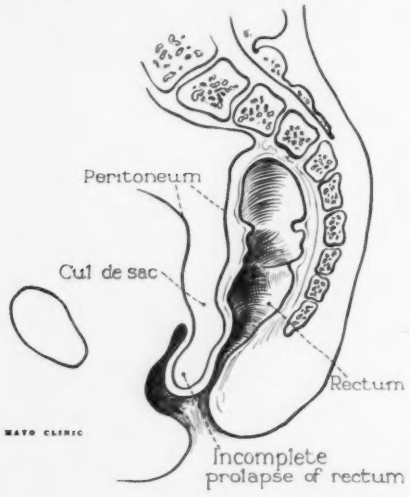

FIG. 2.

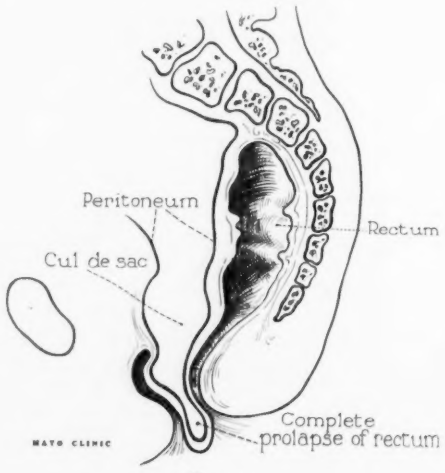

FIG. 3.

FIG. 2.—The basic anatomical changes occurring in prolapse of the rectum (incomplete prolapse). (After Moschcowitz.)
FIG. 3.—Complete prolapse of the rectum. (After Moschcowitz.)

truding bowel retains its normal color and the mucosa has a healthy appearance. Later, due to constant irritation and trauma, the mucosa becomes inflamed and œdematous and frequently constitutes a reddened, oozing mass which may have one or more areas of ulceration. There is usually a constant secretion of mucus which adds to the disagreeable nature of the lesion. As a rule pain is not a prominent feature. In advanced cases, especially if patients are elderly, incapacity and debility are frequently pronounced and often sufficiently severe to confine the patient to bed. Sometimes incarceration or strangulation of the bowel may occur, as in the case of any other hernia.

Treatment.—General hygienic measures are essential in the care of these patients as an adjunct to any form of treatment directed toward effecting cure. The treatment may be medical or surgical. As a rule, medical meas-

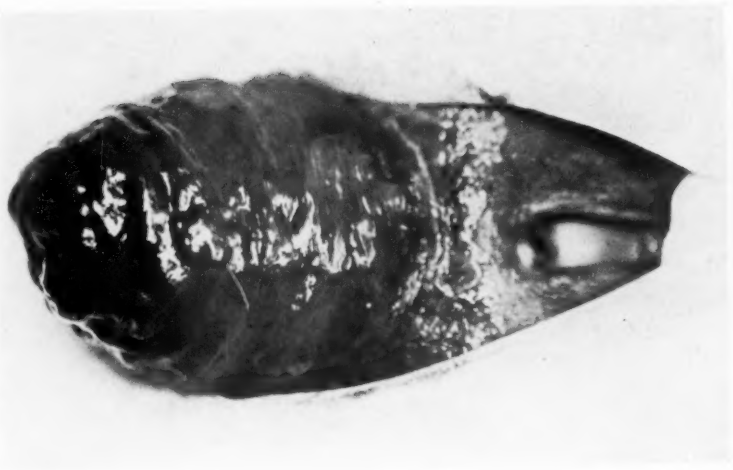

FIG. 4.—Typical clinical picture of prolapse of rectum.

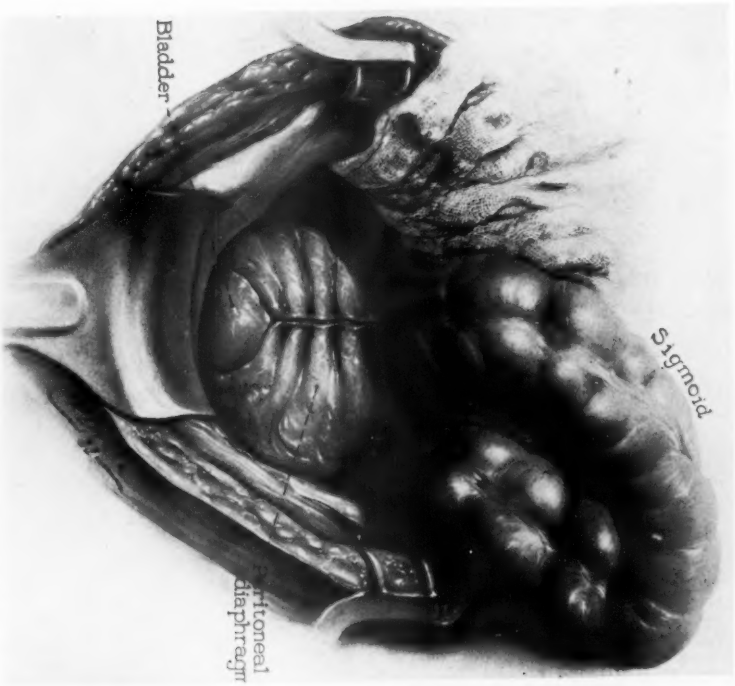

FIG. 5.—Appearance of the reconstructed pelvis following completion of the Moschowitz operation.

ures are effective only in the early cases when the protrusion is small and not associated with advanced, secondary anatomical changes.

Non-operative procedures may be classified as: (1) Soothing ointments or lotions to relieve the irritated mucosa; (2) submucous injections of astringents, such as absolute alcohol, ergot, and so forth; (3) mechanical supports, and (4) electric treatments. It is evident that treatment of this type is more palliative than curative.

Numerous types of operative procedures have been suggested for the cure of rectal prolapse:

(1) Operations for narrowing the anal aperture and adjacent rectum, such as resection of a wedge-shaped segment from the posterior wall of the rectum, cauterization or removal of the rectal mucosa, twisting the rectum, subcutaneous injections of paraffin at the level of the sphincter, and ligation by a loop of silver wire at the level of the anal orifice.

(2) Procedures attempting to strengthen the rectal supports from below, such as plastic operations which utilize the adjacent gluteus muscles, procedures attempting to use the levator ani muscles by suturing them closer together, plastic operations as suggested by Hofmann[1, 2] who made an H-shaped incision posterior to the anus and stretched the side-to-side denudation in an anteroposterior direction and closed the incision with strong, supporting sutures.

(3) Various types of fixation operations, such as rectopexy of the Verneuil[5] type, which consists of removal of the coccyx and suture of the rectum to the integument, and sigmoidopexy, in which the sigmoid is sutured to the anterior abdominal wall; there have been numerous modifications of these various procedures. In general, rectopexy is usually unsatisfactory, as it fixes mainly the posterior wall of the rectum which plays but a small part in the production of prolapse. Sigmoidopexy, with fixation to the anterior abdominal wall, has been received with much favor in the past. This operation is easily performed and in mild cases gives satisfactory results. We have abandoned this procedure, however, because it appears dangerous in certain cases and, furthermore, because recurrence will invariably develop if the prolapse is severe.

(4) Operations directed toward cure by resection of the prolapse may be done with a ligature, an elastic bandage, or by amputation with a knife. The outer and inner cylinders are then joined by suture at the line of section. The results of this type of operation have not been as satisfactory as might be expected. As a rule, these procedures have little in their favor. Resection may become necessary, however, if the prolapse becomes incarcerated or gangrenous.

(5) An operation suggested by Moschcowitz, in which the cul-de-sac of Douglas is obliterated, is based on the theory that true rectal prolapse is a hernia of the sliding type. We believe this supposition to be fundamentally sound and anatomically accurate. The Moschcowitz operation has afforded very satisfactory results in our hands. In the last five years it has been em-

ployed in nine cases of major prolapse of the rectum. The results have been highly satisfactory in seven of these cases. Many of the patients had undergone numerous types of treatment elsewhere before this operation was performed at the clinic.

The principles underlying the technic of the Moschcowitz operation are relatively simple and easy of application. The operation is uniformly applicable in both sexes. It is somewhat more difficult to perform it on men because of the smaller and deeper pelvis. The true pelvis is largely obliterated and a new pelvic floor is formed. By placing row on row of silk or linen purse-string sutures around the cul-de-sac, the operation is started at the bottom of the pelvis and carried upward in subsequent layers several centimetres apart until the entire pelvis is obliterated. Fig. 5 illustrates the appearance of the pelvis at the conclusion of the operation. Care should be employed in placing the sutures to avoid the ureters and internal iliac vessels. One should incorporate a certain amount of subperitoneal tissue in the suture line to strengthen the line of closure. A convenient procedure may be followed by placing all of the sutures before any of them are tied and then proceeding from below upward, tying them in order. It is very important to insert a large-sized rectal tube into the lower part of the bowel as far up as the sigmoid to obviate the possibility of constricting the rectum during the operation. This tube is anchored with a silkworm stitch placed through the anus. It is allowed to remain in place for six to eight days after operation. If women have passed the menopause, ventral fixation of the uterus may add to the success of the operation; if they are younger it is important to remember that parturition almost inevitably leads to a recurrence. Fixation of the sigmoid to the anterior abdominal wall, which has been recommended, is not considered advisable.

After operation, it is desirable to place the patient on a low-residue diet and administer mineral oil daily for a period of at least eight days, when the rectal tube is removed. The diet may gradually be increased and daily enemas of oil given. The patient should remain in bed for two or three weeks following the operation.

Intussusception of the Rectosigmoid.—Intussusception of the rectosigmoid, frequently referred to as a third-degree prolapse, is, strictly speaking, not a true prolapse, but an intussusception of the lower segment of sigmoid and rectosigmoid into the ampullary portion of the rectum. From a clinical point of view, however, these cases have many features in common with those of true major prolapse of the rectum. Etiologically and symptomatically they are similar. Surgically, they are best treated by obliteration of the true pelvis, after the technic described by Moschcowitz. The diagnosis is readily determined by digital or proctoscopical examination. Unlike intussusception in other portions of the large bowel, strangulation, complete obstruction or adhesions of the invaginated surfaces rarely occur. Depending on the extent of the lesion, the bowel may or may not protrude

through the anus. A case, the history of which follows, is illustrative of this type of lesion.

A physician, aged forty-three years, first examined at The Mayo Clinic October 10, 1931, complained of a "dragging down" sensation in the lower part of the abdomen and a relaxed anal aperture. Constipation was severe enough to require a daily enema. The patient complained of severe exhaustion which he attributed entirely to the rectal disorder. In other respects his general health was good and he had not lost weight.

The patient weighed 147 pounds. The results of general examination and of laboratory tests were all negative. Proctoscopical examination disclosed a mobile and redundant rectosigmoid which prolapsed down into the rectum. A diagnosis of prolapse of the rectosigmoid was made.

October 23 a Moschcowitz type of operation was performed. The tissues of the pelvis were found to be extremely relaxed and redundant and the true pelvis was obliterated. The immediate post-operative convalescence was uneventful and the patient left the hospital two weeks after operation. When last heard from, six months afterward, he was well and completely relieved of all previous rectal complaints.

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PRURITUS ANI: ITS ETIOLOGY AND TREATMENT *

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A survey of the literature on pruritus ani leads to the conviction that the etiology of this very prevalent complaint is not understood and that its treatment is quite unsatisfactory. A recent study of this condition at the Stanford Clinic has made possible certain definite conclusions as to its etiology and has led to more satisfactory results in its treatment.

Pruritus ani is the *symptom* of localized itching about the anus. It is not a clinical entity, always having the same fundamental cause, and the search for causative factors has led certain investigators far astray. A brief summary of the many and varied theories of etiology seems desirable before presenting the writer's understanding of the condition.

Perhaps the earliest conception of etiology was that of an hysterical or neurogenical background as the fundamental trouble. Even as recently as 1926, Rice[1] believed that there was little doubt that pruritus ani "is largely influenced by an inherited or acquired neurotic condition." In 1929, Smith[2] felt that in many cases it "is an outlet for emotional stress." Beach[3] propounded the interesting hypothesis that "periods of financial depression are not an unlikely factor."

Innumerable constitutional disturbances have been held responsible, as, for example, gout, diabetes, rheumatism, lithæmia, uræmia, hepatitis, nephritis, syphilis, tuberculosis, malaria, glycosuria, typhoid disturbance, and ovarian dysfunction. Generalized itching does occur with at least some of these constitutional diseases. But this must not be confused with the condition under discussion, which is that of localized itching in the anal region. Lockhart-Mummery,[4] in a discussion of diabetes as the possible cause of pruritus ani, states that he has never seen such a case. The unanimous opinion of many colleagues consulted denies the association of localized perianal itching with any of these conditions.

Another somewhat fanciful theory is that propounded by Clemons,[5] who believes that pyorrhœa alveolaris is the underlying source of the trouble. Kiger[6] resorts to that tottering concept of a distal focus of infection, naming specifically tonsillitis, alveolar abscess, sinusitis, chronic appendicitis, and cholecystitis.

There has been an increasing trend in support of the theory that pruritus ani is a referred sensation caused by disease in some distant organ. At first contiguous organs, such as the prostate, the seminal vesicles, the bladder, and

* Read before the Surgical Section, San Francisco County Medical Society, August 16, 1932.

the urethra were implicated. The indictment was later broadened to include all the abdominal viscera.

Pain as a referred symptom of disease occurs frequently and by a well-understood mechanism. But itching is never encountered elsewhere in the body as a referred sensation. The pain of biliary colic often radiates through to the back and to the right shoulder blade. But it never occurs at the anus! If itching were to occur as a reflex symptom of gall-stones, it should occur over the right shoulder blade, not at the anus. The pain of renal colic may radiate along the course of the ilio-inguinal nerve through the groin to the scrotum and to the inner side of the upper thigh. It never occurs at the anus. If itching were to occur as a reflex symptom of renal colic it should occur in this same region and not at the anal canal.

It is difficult to accept the theory of reflex origin as reasonable, nor has clinical experience necessitated recourse to this ill-conceived hypothesis.

Attention has been so focused upon the remote causes of pruritus ani that local causes have been neglected and even when obviously present have been treated with contempt. Proper investigation of possible local causes requires special instruments, technic, and experience. Cursory external inspection and the blind insertion of a gloved finger into the rectum is more often than not totally inadequate. Direct visual inspection of the entire length of the anal canal, the mucocutaneous line, and at least the lower two inches of the rectum, by means of a properly devised and adequately lighted anoscope, is imperative. Only then can one obtain a thorough understanding of the local mechanical and infectious factors involved. *Experience has shown that there is always a local cause for the symptom of localized anal itching.*

Crookall,[7] in 1912, pointed out an important fact in relation to this condition. The entire length of the anal canal up to the mucocutaneous line at the upper end is lined with stratified squamous epithelium which is notably lacking in the usual horny layer to which the skin elsewhere in the body owes its impermeability and in the fibrous tissue of the reticular layer to which the skin elsewhere owes its toughness. Because of this fact, maceration, erosion, and splitting of the anal skin are easily produced by factors which elsewhere in the body would have no effect, with a consequent irritation of the underlying nerve-bulbs.

The essential factor in the production of itching is the abnormal presence in the anal canal of an irritative secretion or discharge. A number of conditions may be responsible for this. The first group to consider includes those conditions which allow an abnormal seepage of the ordinary secretions of the rectal mucosa into the anal canal. Normally, this canal is held firmly collapsed throughout its entire length by the encircling sphincter muscles, with the firmest occlusion at the level of the upper end of the canal. This prevents any leakage of rectal contents into the anal canal except at moments of defecation. Any mechanical interference with the normal closure of the canal may allow constant leakage with a resultant maceration and irritation of the wall, inciting itching. This may be caused by a relaxed sphincter muscle,

either neurogenical or post-operative. Internal hæmorrhoids may prolapse far enough into the anal canal to prevent proper closure, allowing leakage, even though the prolapse is not far enough to be appreciated by the patient. Hæmorrhoidal masses beneath the skin of the upper end of the canal may have the same effect. These are really external hæmorrhoids, being covered by stratified squamous epithelium, although there may be no evidence of their presence on external examination. A hypertrophied papilla may be responsible.

In addition to these mechanical factors, infectious discharges may also produce irritation of the anal canal. Pockets with inadequate external drainage may develop above hæmorrhoids or in enlarged crypts. Erosion of the delicate mucous membrane by some minute foreign body may occur, with the development of a low-grade infection. In the case of hæmorrhoids, inadequate closure of the anal canal may allow the irritative discharge continually to invade the canal. In cryptitis, infection often burrows beneath the anal skin, which becomes friable and may split with the formation of a small fissure or sinus tract. Here, again, an irritative discharge has continual access to the anal canal.

A specific infection of the skin of the anal canal by streptococcus fecalis was advanced by Murray[8] as the etiological cause of pruritus ani. While this was momentarily hailed as a great discovery, subsequent work by Montague[9] and others failed to confirm any specific relationship. Montague was also able to show by a careful study of microscopical sections of anal skin from patients with pruritus ani that while occasional organisms were found in the superficial eroded layers of the skin, there was no appreciable bacterial invasion.

The possibility of a mycotic infection as the causative agent was first mentioned by Ball.[10] Several casual references to this as an occasional cause are found in the more recent literature. The only available studies from this viewpoint are those of Castellani[11] and of Terrell and Shaw.[12] In 1925, Castellani reported careful mycotological studies on all cases during a period of thirteen years, 20 per cent. of which showed epidermophytons present in scrapings made from the anal or perianal skin. Terrell and Shaw, in 1928, confirmed Castellani's work, but their results are rather indefinite.

For the past year, Doctor Chope, of the department of bacteriology, has undertaken mycotological studies in those patients in which we suspected the possibility of a mycotic infection. The suspected cases showed a characteristic picture. The skin was gray and sodden in appearance, with areas of erosion hidden in the crevices of deep longitudinal folds. The skin was so thickened and rigid that when the buttocks were pulled apart the anal canal appeared funnel-shaped. In contrast to this type of case is that in which there is little evidence of thickening of the skin, the surface is smooth and moist, often with weeping excoriations resulting from scratching.

A fungus infection was suspected in eleven cases. In three of these direct smears made from scrapings taken from areas of erosion showed branching

septate mycelia. In no case was it possible to culture a fungus on Sabouraud's medium. Treatment with a fungicide improved the appearance of the anal and perianal skin, with healing of ulcerations, softening of the skin, and a return to fairly normal appearance. However, complete relief from pruritus did not result in a single instance. In every case pathological conditions such as those described as primary were found. Nine of these patients have been cured completely by treatment of these underlying conditions. The other two have not as yet undergone treatment. This rather small series suggests that secondary infection by fungi may occur in the moist, macerated skin resulting from a chronic, irritative discharge, but that it is not the fundamental cause of the itching.

Treatment.—Methods of treatment are as myriad as the hypotheses of origin. Being convinced of the universal presence of a local cause, I am equally convinced that the only logical and satisfactory treatment for obtaining permanent relief must be that directed toward the cure of the local cause.

Removal of the prostate, the ovaries, the uterus, the appendix, the gallbladder, or the teeth is based upon a fallacious premise and is useless. All of these procedures have been advocated and performed.

There are many types of therapy directed at the secondary changes which occur in the anal skin, without endeavoring to remove the primary cause. To be sure, some measure of relief is sometimes obtained by such methods of treatment, but the results are neither satisfactory nor permanent. Radiation is one such form of therapy. Extensive burns following such treatment resulting in years of suffering present convincing evidence that radiation gives little relief and may do great harm. While ultra-violet light may toughen skin and inhibit bacterial growth, this form of therapy does not strike at the true causative factors. Ionization therapy was inaugurated in 1921 by Rolfe,[13] in an endeavor to attack the bacteria supposedly buried deep in the anal skin. His average course of treatment was three times weekly for five months. Published results show some relief in 50 per cent. of his cases.

The utilization of specific and non-specific vaccines has been advocated by many authorities following the successes reported by Murray, in 1911. In 1924, Montague[9] concluded that it was "totally unsatisfactory." Its status is the same today. Hazen,[14] in 1923, maintained that what success had been obtained by vaccine treatment probably was the result of protein shock. The latter has not been proven of any value.

Jamison,[15] in 1918, advocated colonic flushes at temperatures of 125–150° F., claiming that hot water is antiphlogistic, antispasmodic, antiseptic, antacid, antiflatulent and anodyne, its action softening indurated and cicatricial tissue, equalizing the circulation, and stimulating secretion and excretion. In short, it would appear to do everything but cure the itching.

Numerous methods have been devised for blocking the sensory nerve endings of the anal and perianal skin. Complete relief may be obtained by some of these methods for from three to twelve months, but, unless the

causative pathological condition is rectified, this is not a permanent cure. Probably the first procedure of this type was the undercutting operation devised by Sir Charles Ball.[10] Variations of this operation have been offered from time to time by Mathews,[16] Allen,[17] Hanes,[18] and others. A much simpler and equally effective means of blocking the sensory nerves is by subcutaneous injection of some one of various sclerosing or anæsthetizing solutions. Stone[19] employs 95 per cent. alcohol, Goldbacher[20] advised 5 per cent. phenolized oil, Yeomans[21] has produced benacol, and Gabriel,[22] A.B.A. solution. Hanes[23] has found 1:3000 hydrochloric acid satisfactory. The solution of choice is probably one-third of 1 per cent. quinine and urea hydrochloride as advocated by Yeomans, which can be injected in considerable quantities without the danger of sloughing apt to occur with certain of the other solutions mentioned. The addition of enough novocaine to make a 1/8 per cent. solution is sufficient to control the rather severe burning experienced during the actual injection of the quinine and urea hydrochloride. Immediate relief is usually experienced, and may last from three to twelve months. Permanent cure is never expected.

It would be useless to list the innumerable salves and ointments that have been advocated. They are, at best, palliative, and do not reach the fundamental trouble.

In my own treatment of these patients, a meticulous visual examination of the entire length of the anal canal and lower rectum is made for the discovery of the possible cause of an abnormal irritative discharge. Treatment is then instituted for the correction of the local lesion. In the majority of cases surgical correction is necessary, although in some cases the injection of internal hæmorrhoids or the non-surgical treatment of a cryptitis may be adequate. In certain cases, where the patient is not willing to await the healing of the operative wound for relief, subcutaneous injection of one-third of 1 per cent. of quinine and urea hydrochloride may be employed for immediate relief, depending on correction of the pathological condition for permanent cure.

In 304 patients seen in the proctological clinic from September, 1931, to July, 1932, 152, or exactly 50 per cent., have suffered from localized anal itching. Of the other 152 patients, twenty-nine were seen for various disturbances not involving the anorectal region, which was found to be normal. Therefore, of 275 patients with anorectal disease, 54 per cent. complained of anal itching. This serves to point out the frequency of this symptom in association with anorectal disease. A definite local cause was found to account for the pruritus in every one of the 152 cases except the very first case registered in the clinic. Inexperience and momentary enthusiasm for the mycotic theory of etiology resulted in failure to recognize the true local cause. A desire to check up on this patient has been thwarted by her return to Germany.

Space does not permit consideration of the detailed findings in each of the

152 cases which are contained in this record. The various local causes found include all those previously described.

Treatment was undertaken in only sixty-three of the 152 cases. The majority of those not having treatment were unable to obtain the funds requisite for a few days' stay in the hospital for necessary operative procedures. Four who were told that operative measures would be necessary for cure were given palliative treatment by injection of hæmorrhoids because of their inability to finance an operation. All four were markedly improved after the injection of large eroded internal hæmorrhoids. Complete relief in these patients depends, however, upon the correction of mechanical factors not amenable to injection therapy. Two patients had eroded hæmorrhoidal masses located entirely above the mucocutaneous line, which were prolapsing into the upper end of the anal canal. Injection treatment gave these patients complete relief for three and eight months. Their hæmorrhoids then recurred, as did their pruritus. Both patients had been told at the time of their original treatment that injection was not a cure for their hæmorrhoids. Both are now anxious to obtain more permanent relief by hæmorrhoidectomy. Two patients were treated for a considerable length of time on the supposition that mycotic infection was responsible, definite local pathological conditions within the anal canal being found at the original examination but ignored. Treatment was not satisfactory, and the necessary operative measures have not yet been undertaken. This leaves fifty-two patients in whom treatment has been completed. Of these, fifty-two, or 100 per cent., have enjoyed complete and absolute relief from their pruritus, without recurrence, and without the use of analgesic ointments or the employment of the various nerve-cutting or nerve-blocking procedures which may give temporary relief. The one exception to this was a man in whom subcutaneous injection of one-third of 1 per cent. quinine and urea hydrochloride was done to give immediate relief for a very severe itching. His hæmorrhoids were then treated. The relatively brief period of relief in many of these cases prohibits dogmatic assertion of permanent cure, but their relief should endure just as long as there is no recurrence of the original local pathological condition.

In summary, our experience would seem to justify the statement that for every case of pruritus ani there is a definite local cause, detectable by proper examination, and amenable to proper treatment.

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NON-SPECIFIC GRANULOMATA OF THE INTESTINES*

(INFLAMMATORY TUMORS AND STRICTURES OF THE BOWEL)

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During the past few years we have encountered cases in increasing frequency which clinically and radiologically gave the impression of tumor or tuberculosis of the bowel. The appearance of the bowel at operation likewise was usually considered to be either that of hyperplastic tuberculosis or of malignant disease. Examination of the resected specimens, however, failed to substantiate such a view. No evidence of specific disease, such as tuberculosis, syphilis or actinomycosis could be found. Amœbic disease of the bowel was excluded on study of the microscopical sections and of the stool, also by the inefficacy of emetin therapy in suspected cases. Carcinoma, lymphosarcoma, and Hodgkin's disease were definitely excluded. In a few of these cases the condition was evidently secondary to diverticulitis. Aside from these, a large heterogeneous group of cases remained, differing from one another etiologically, but with certain common clinical and pathological findings. These cases, showing various degrees of chronic productive inflammation in different stages of healing, have long been known to English as well as continental surgeons. In 1921, Tietze[1] published a thorough résumé of the subject with a very complete bibliography. In 1923, Wilensky and Moschcowitz[2] reported four cases collected from various institutions under the designation "Non-Specific Granuloma of the Intestine," perhaps the most useful classification from the standpoint of the underlying pathology. Mock[3] recently reported a series of cases using practically the same designation. Clinically, these cases manifest themselves either by the development of palpable masses or by symptoms pointing to stricture of the bowel with ulceration. They may, therefore, with propriety, also be designated as non-specific inflammatory tumors and strictures of the bowel.

Both the intestine and its peritoneal covering are known to possess remarkable powers of resistance to infection and inflammatory lesions within them show a striking tendency to undergo resolution. Moreover, the intestinal mucosa in itself possesses marked regenerative power.[4] Surgeons have frequently recorded the amazing rapidity and completeness with which huge inflammatory exudates and masses may disappear from the abdomen. Very extensive disease or injury of the mucosa may heal without permanent scarring resulting. In some instances, however, following infection or injury, *restitutio ad integram* does not occur. The persistence of infection or other

* Presented before the American Gastro-Enterological Association in May, 1932. The section on Localized Ileitis represents a joint study with Dr. Burrill B. Crohn.

irritating factor or the inability of the tissues to overcome them, results in a series of reparative and destructive processes which lead ultimately to the formation of either hypertrophic peri-intestinal masses or extensive intramural hypertrophic ulcerative and stenosing lesions or combinations of both.

Histological study of the various types of lesions shows merely evidences of various degrees and stages of acute and chronic inflammation with infiltration by lymphocytes, polymorphonuclear leucocytes, plasma cells and mast cells together with varying degrees of fibroblastic proliferation and often degenerative changes. In some of the peri-intestinal lesions there is often considerable hyalinization and calcification and even early bone formation has been encountered.

The presence of giant cells is a common finding. We believe that these are of only accidental significance and are due to the presence of particles of non-absorbable vegetable matter which have become incorporated within the lesion as a result of deep ulceration, remaining entrapped within the healing tissues. Wilensky and Moschcowitz reported the presence of certain large cells in the microscopical sections of one of their cases. By means of special methods of staining, Doctor Antopol has been able to demonstrate in a few of our cases cells or groups of cells which are, in all probability, vegetable cells and around which giant cells are usually present. We have found them in the serous and sub-serous layers of the bowel, as well as within the submucosa, which may be an indication of their power to traverse the bowel wall through lymphatic channels. Being non-absorbable, they become encapsulated by foreign body gaint cells. In the serosa they give rise to little nodules which can be differentiated grossly with great difficulty from miliary tubercles. Similarly foreign body tubercles have been produced experimentally by the introduction of emulsions containing vegetable matter into the peritoneal cavity.[5, 6, 9] The presence of vegetable cells is of two-fold importance; by their irritant action they may be a contributing factor in producing the marked hyperplastic fibrosis seen in some cases; in addition, they are probably responsible for the confusion of these non-specific lesions with tuberculosis, due to their stimulating the production of giant cells.

The following is based on a study of fifty-two cases which have been observed and operated upon, most of them at the Mount Sinai Hospital in the Surgical Service of Dr. A. A. Berg during the past ten years. Only cases* in which resection was performed or specimens obtained are considered. The microscopical sections were restudied with the invaluable aid of Dr. Paul Klemperer in order to settle certain questionable points. An accurate etiological or pathological classification is at present impossible. We submit the following classification, therefore, fully conscious of its defects and overlappings, but pleading in its favor a certain degree of clinical utility.

* Cases of sigmoid diverticulitis or cases with lesions situated distally to the recto-sigmoid junction are not included. Two cases of ulcerative jejunitis near the fossa of Treitz have also been excluded from this study.

It is our plan to discuss each group and report in short abstracts some of the typical cases.

(1) Extra or peri-intestinal granulomata secondary to sealed-off perforations of the bowel.

(2) Granulomata secondary to known vascular disturbances of the gut.

(3) Localized hypertrophic ulcerative stenosis of the terminal ileum. (Regional ileitis.)

(4) Localized hypertrophic colitis with or without low-grade generalized colitis.

(5) Simple penetrating ulcers of the colon.

(6) Lesions secondary to inflammation of the appendages of the bowel such as appendicitis, diverticulitis, typhlitis.

(I) LESIONS IN WHICH THE INFLAMMATORY REACTION IS MAINLY EXTRA- OR PERI-INTESTINAL AND WHICH ARE SECONDARY TO SEALED-OFF PERFORATIONS OF THE BOWEL

As a response to a slowly perforating lesion of the intestine that has become sealed off by omental, parietal or visceral adhesions, large inflammatory masses with very little or no pus formation may develop. These are intimately adherent to the serosa and subserosal tissues but do not actually involve the submucosal and muscular layers of the gut. A typical example is the lesion resulting from perforation of the colon by foreign bodies, such as fishbones. Usually, this accident results in the formation of a localized intra-abdominal abscess. In some instances, however, possibly due to a slow rate of perforation, the inflammatory reaction is chiefly productive. As a result of the continued presence of a penetrating foreign body and the low-grade infection resulting a marked hypertrophic inflammatory reaction takes place in the pericolonic and subserous layers of tissue and both clinically and at the operating table may give the impression of being a colonic neoplasm. Three of our cases were operated upon with a previous clinical diagnosis of malignant neoplasm but were recognized as inflammatory tumors at the time of operation and foreign bodies sought for and found. Perforative lesions, from whatever cause, may involve the omentum with the development of a large omental mass of tissue with areas of necrosis and xanthomatous change, firmer fibrotic masses, areas of calcification or discrete encapsulated miliary abscesses. The lesion in the wall of the gut may be minimal.

An exceedingly interesting group are those cases in which a pseudo-tumor of the abdomen wall itself develops as a result of a perforative lesion becoming sealed off by the anterior parietes. In two such cases, foreign bodies (fish-bone, toothpick) were found in the centre of a large firm tumor mass involving the rectus muscle and pro-peritoneal tissue. Each of these tumors had been excised because of a diagnosis of sarcoma of the abdominal wall. The opposite surface of each of these masses was densely adherent to the omentum but there was no evidence of adherent bowel. In two other

cases in which the same pre-operative diagnosis of sarcoma was made adherent gut was found on the deep side of the mass, a perforation apparently having been sealed off by the parietal peritoneum. The "tumors" were found to be œdematous, granulomatous lesions involving the parietal peritoneum and the pro-peritoneal cellular tissue.

A total of ten cases* makes up this group, five of them definitely due to foreign body. In three cases, the inflammatory mass was mainly in the serosal and subserosal layers of one of the flexures of the colon. In two instances the mass was mainly omental, the original site of the perforation being in the transverse colon in one instance and in the small intestine in the other. In four cases the main mass of granulomatous tissue lay in the abdominal parietes. In one instance it was chiefly perivesical.

Clinically, these cases were characterized by the development of a palpable mass without symptoms of obstruction or ulceration. Because of the lack of intramural involvement of the bowel, the barium meal or enema usually showed no abnormalities. Occasionally, a persistent spasm of the bowel adjacent to the lesion was encountered and gave the impression of a filling defect. At operation these tumors were not found to encroach upon the intestinal lumen, and intra-intestinal irregularities or ulcerations were absent. This point is of use in differentiating densely adherent peri-intestinal inflammatory masses from neoplasm or from inflammatory diseases which actually involve the bowel wall.

(II) LESIONS SECONDARY TO KNOWN VASCULAR DISTURBANCES OF THE BOWEL

The most striking example of this type of lesion that we have encountered is the stenotic involvement of the bowel which occurs when badly compromised but viable gut has been replaced following operation for strangulated hernia.[11] In these cases there is permanent vascular injury affecting the intramural vessels of the bowel. Vascularization through collateral anastomoses is sufficient to prevent necrosis of the muscular and fibrous layers of the bowel. It is, however, insufficient to permit the usual and very active regeneration of the mucosa. Ulcerative lesions of the mucous membrane ensue followed by secondary infection and the gradual production of severe grades of cicatricial stenosis of the intestinal lumen. Five such cases have been encountered in this series. All were of the type of a tubular stenosis, *i.e.*, stenosis involving an entire segment of bowel corresponding to the extent of previous strangulation. Cases of narrow annular stricture corresponding to the site of constriction at the neck of a hernial sac have been reported in the literature. In this series we observed one annular stricture in which a loop of gut had been caught under an encircling band of omentum. Small mesen-

* The large group of cases with marked productive pericecal changes due to appendicitis and perisigmoidal inflammatory masses from sigmoid diverticulitis also belong in this group and are much more common than any of the other types of lesions noted. They are so well known, however, that they have not been especially studied or included in the enumeration of this group.

teric tears have also been reported as causing similar stenosis and through a like mechanism.

The symptoms were those of gradually increasing subacute intestinal obstruction; the ulcerative phase apparently passed by unnoticed. The symptoms of obstruction appeared anywhere from two weeks to six months following the strangulation. Short-circuiting entero-anastomosis afforded relief of symptoms in four cases, resection in one. One patient came to autopsy without operation.

In these cases we have definite evidence from the history and findings at the primary operation that there had been an extensive vascular insult. How large a rôle the vascular mechanism plays in cases where the causal connection is not so clear cut is difficult to say. Although not susceptible of proof, the possibility must be borne in mind that such conditions as repeated, self-reducing intussusceptions or recurrent partial volvulus especially at the ileocecal angle may be responsible for certain chronic inflammatory lesions. It must be emphasized that the end stages of a lesion in which primary vascular insufficiency produced necrosis and secondary infection of the bowel resemble in most respects those in which a primary infectious agent has produced secondary and thrombotic and degenerative changes.

(III) HYPERTROPHIC ULCERATIVE STENOSIS OF THE TERMINAL ILEUM. (Regional ileitis)

In this group of cases the terminal ileum was the seat of the lesion which was especially marked at the ileocecal valve, usually terminating abruptly on the ileal side of the valve. Proximally, the lesion diminished in severity, the signs of the disease being rarely found farther than twelve to fifteen inches from the ileocecal junction. We have no clue to its etiology but observation of various stages of the disease process in different patients leads us to believe that the following are the steps in its development. The primary stage, we believe, occurs in the form of multiple oval or lenticular ulcerations in the mucosa of the mesenteric side of the bowel. We have found this lesion on a number of occasions proximal to the main hypertrophic mass and separated from it by normal appearing mucosa. As the disease progresses it is characterized by two main features; first, a marked tendency to perforation, and second, an excessive proliferative reaction in the submucosa. The end stage of the process, the one most frequently encountered, is manifested by the conversion of the terminal ileum into a thickened, rigid, hose-like tube. (Figs. 1 and 2.) When opened, the normal transverse intestinal folds in the terminal portion of the ileum are seen to be in part destroyed and in part flattened and broken up into polypoid masses. A row of linear ulcerations in the mucosa overlying the mesenteric border is practically a constant finding. In places, especially near the cæcum, the mucosa is almost completely atrophic and there may be papillary excrescences, especially along the margins of the ileocecal valve. The submucosa is enormously thickened and contributes to the marked diminution in the calibre of the lumen. Perforation

may occur after adhesion of two loops of bowel or between the leaves of the mesentery, which with the enlarged glands gives rise to fairly large masses. The purulent exudate may force its way through loose cellular tissues forming secondary fistulous communications with the cæcum or colon. At times the perforation occurs into the peritoneal cavity with formation of an intraperitoneal abscess. Drainage of these abscesses results in the formation of intractable fistulæ. These patients present themselves in four different clinical pictures.

(1) *Symptoms Simulating Those of Acute Appendicitis.*—The first sign of the disease may be an attack which is impossible to differentiate clinically from appendicitis. At operation, however, it is at once noted that the terminal

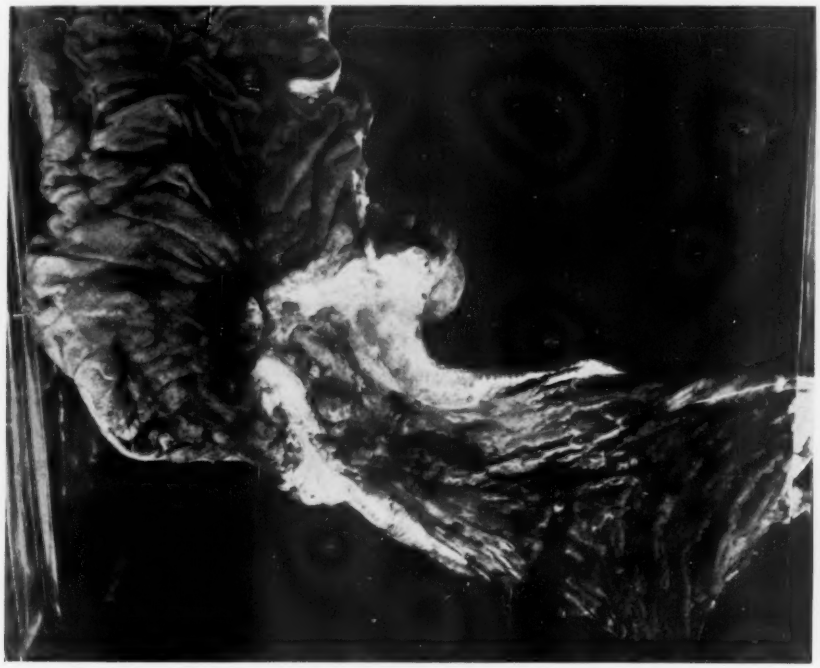

FIG. 1.—Non-specific inflammatory stenosis of the terminal ileum. The inflammatory process ends rather abruptly at the ileocecal valve. The fibrotic thickening of the ileal wall is well shown, as are some polypoid mucosal masses.

ileum is soggy, œdematous and blotchy, and that there are numerous large succulent glands in the terminal mesentery. In no case of our series was resection performed in this stage of the disease so that we are ignorant of the underlying pathological changes present during this stage. A number of these patients were observed clinically as they passed into the more chronic phases of the disease. One case is symptom-free two years after exploration although X-ray shows definite narrowing in the terminal ileum.*

Occasionally, these patients are admitted with an abscess already present. Drainage may result in the formation of a chronic fistula immediately after

* Since the completion of this article the patient has reappeared with recurrence of symptoms. Resection revealed a typical hypertrophic ileitis.

operation or there may be primary healing with secondary breaking down, occurring weeks or even months later.

(2) *Symptoms of Ulcerative Enteritis.*—There may be a low-grade diarrhœa, loss of flesh and strength, mild colicky pains and development of a secondary anæmia. This type is rather uncommon.

(3) *Symptoms of Chronic Incomplete Obstruction of the Small Intestine.*—This is the most common manifestation of the disease. The patient may have previously passed through one of the phases described above, but frequently the symptoms of obstruction appear without any previous history. Severe abdominal cramps, visible peristalsis, and borborygmi are the symptoms complained of most frequently. The duration of symptoms in this

FIG. 2.—Non-specific hypertrophic stenosis of the terminal ileum. The gradual transition from normal bowel to the extensively diseased portion is well shown. The probes pass into perforations on the mesenteric side of the bowel, which formed abscesses in terminal mesentery. At the hepatic flexure is the site of a perforation into the colon.

group varies from one to three years. At operation there is found the characteristic hose-like appearance of the ileum referred to above, frequently very densely adherent to or communicating with the cæcum, ascending colon or sigmoid. The occasional presence of small tubercle-like nodules may render differentiation from tuberculosis difficult. The nature of these foreign body tubercles has been discussed above.

A mass was palpable in every case of subacute or chronic type observed by us. Visible peristalsis was frequently noted. The barium enema, as a rule, showed no abnormality since the disease extends no farther than the ileocecal valve. However, pathological ileocolic or ileosigmoidal secondary communications may be formed and be reflected in the barium enema; this may lead to a false conception of the nature of the disease process. (Fig. 3.)

FIG. 3.—Barium enema showing irregularity near the hepatic flexure in a case of hypertrophic ileitis perforation into the colon. In such a case the X-ray may be misleading and may give rise to the diagnosis of primary colonic disease.

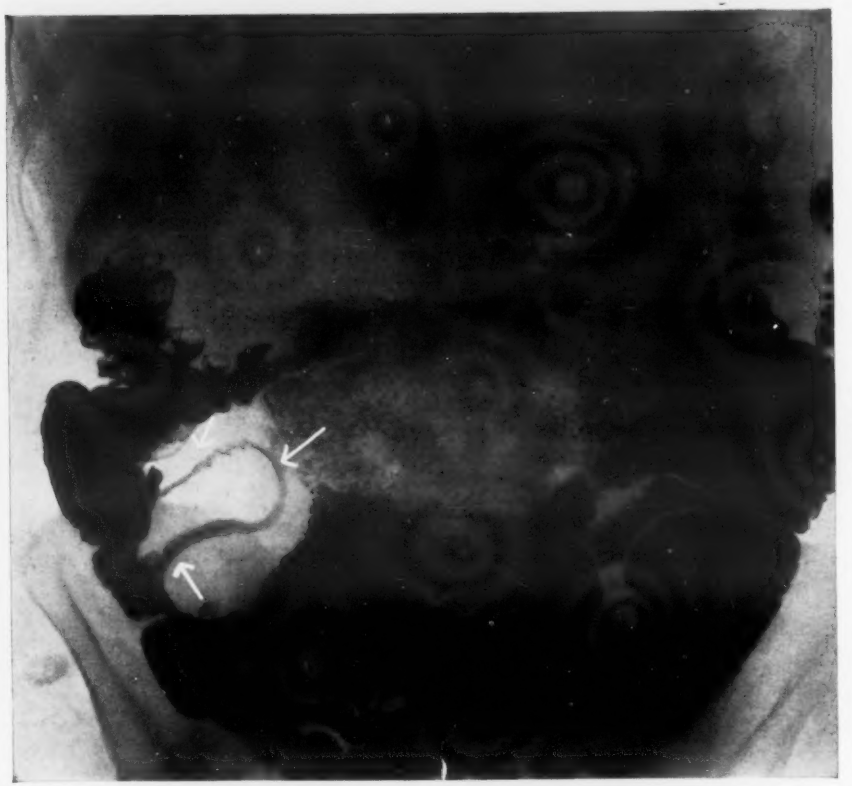

FIG. 4.—Barium meal in a case of hypertrophic inflammation of the terminal ileum. Diseased portion appears as narrow cord-like structure, indicated by the arrows.

In two instances, barium meal demonstrated definite narrowing in the terminal ileum and ileal stasis. (Fig. 4.) In most cases, however, no attempt was made to administer a barium meal because of the fear of precipitating complete obstruction.

(4) *Chronic Intractable Fistulæ.*—These have resulted following drainage of intra-abdominal abscesses and have resisted attempts to produce closure by exposure of the internal opening and simple suture with enterostomy. Cure has been achieved either by short-circuiting operations with exclusion of the involved loop or by resection of the diseased segment with enterocolostomy. The findings at operation are those described under the chronic form of the disease with the added presence of extensive adhesions.

There have been thirteen ileocecal resections in this group with one death. Of the surviving twelve patients, one returned with an annular stricture a few inches proximal to the site of the original resection. The other cases have done well. We have only one *proved* case in which a previous short-circuiting operation had been performed, this patient later requiring resection. An anastomosis had been made, apparently through diseased tissue, without division of the afferent ileal loop, with resultant implantation of the disease on the colonic side of the anastomosis. There were four other patients, who, we believe, fall into this group in whom enterocolostomy with exclusion proved curative. However, no specimens were removed from these patients and they are not definitely included in this series.

Relation to Appendicitis.—We have not been able to establish a relationship between appendicitis and the condition found in any of our cases. Approximately half of these patients had been subjected to previous appendicectomies. In some of them it had been noted at the time of the appendicectomy that distinct abnormalities were already present in the terminal ileum. In the other patients the appendix was examined at the time of resection but no intrinsic abnormalities were found in it, except a severe peri-appendicitis.

Relation to Tuberculosis.—Careful study of the microscopical sections revealed no definite tubercles and no caseation necrosis, nor could tubercle bacilli be demonstrated. In six instances, guinea-pig inoculation, inoculation into rabbits and into chickens were made and all failed to show evidence of any variety of tuberculosis. Lowenstein cultures for tuberculosis were made in three instances and proved negative.

It might be argued that evidences of tuberculosis would be difficult to find in tissue which has undergone fibrosis. However, neither tubercles nor tubercle bacilli could be demonstrated even in the active ulcerative lesions found proximal to the main hypertrophic mass. Even in the tubercle-like structures occasionally seen on the serosa, evidence of tuberculous infection could not be found.

During the past ten years there have been only six cases of *localized hypertrophic ileocecal tuberculosis* resected surgically at the Mount Sinai Hospital as against eighteen of the non-specific variety.* There have been

* Well-defined active cases of pulmonary tuberculosis are not as a rule admitted to the hospital.

a number of instances of multiple tuberculous foci in the intestines found in the course of laparotomy. There have also been a few cases with active or advanced pulmonary tuberculosis showing X-ray evidence of involvement of the ileocecal region; in such instances operation was never resorted to.

To sum up our impressions of "primary" *localized hyperplastic ileocecal tuberculosis,* we would state that in our experience a surprising number of cases diagnosed as such clinically proved on microscopical examination to be non-tuberculous lesions, while those indubitably tuberculous proved to be unsuitable for operative resection because of the multiplicity of these lesions.

(IV) LOCALIZED HYPERTROPHIC COLITIS

In addition to the localized palpable inflammatory mass, some of the cases at one time or another presented evidence of a low-grade general colitis, although much milder than the usual diffuse ulcerative type. Conversely, ulcerative colitis may affect predominantly or almost exclusively certain segments of gut, a fact which has been emphasized by Doctor Berg for years in discussing such cases on rounds; recently Bargen[7] has reported a series of cases illustrating the same point. In most of the present cases there were no röntgenological or sigmoidoscopical evidences of generalized colitis prior to the time of operation and the symptoms were attributed entirely to the localized colonic disease. In a few of these cases, persistence of symptoms after resection led to renewed investigation which in some instances showed evidences of mild general colitis which responded to medical therapy. In only one case did definite symptoms appear first in relation to a segment of gut other than that in which the hypertrophic mass ultimately developed. (Case II.)

Of this group the cæcum and ascending colon were the seat of the lesion five times; the rectosigmoid three times; the mid-sigmoid once, and the junction of the sigmoid and the descending colon three times. In the ascending colon, the hypertrophic disease usually extended upward until a few inches from the hepatic flexure. The mucosa showed occasionally large irregular ulcerated areas reaching one-half inch in diameter with areas of hypertrophic mucosa between them. (Fig. 5.) In other cases the ulcers were smaller and were overshadowed by the bullous polypoid mucosa. Papillomatous and polypoid changes were common in the mucosa. The submucosa was moderately thickened and œdematous. The serosa was opaque, and there was marked thickening and hypertrophy of the subserosal fat both in the colon and mesocolon. The lymph-nodes in the ileocecal angle were enlarged. Numerous adhesions to the omentum and to surrounding loops of gut were found at operation.

In the sigmoid the pathological changes showed a greater cicatricial tendency with more limited involvement and relatively little ulceration. There was a greater tendency to development of strictures and papillary proliferation in the mucosa. (Fig. 4.) Microscopically, merely various stages of

non-specific inflammation were seen. Careful search was made for amœbæ as well as tubercle bacilli, but no evidence of either was found.

Most of these patients had been ill for about six months before coming for operation. Abdominal pain, diarrhœa, and bloody stools were the most common complaints when the proximal colon was involved. When the sigmoid was involved, constipation and painful defecation were present. In some the symptoms were mainly those of obstruction. A mass was found

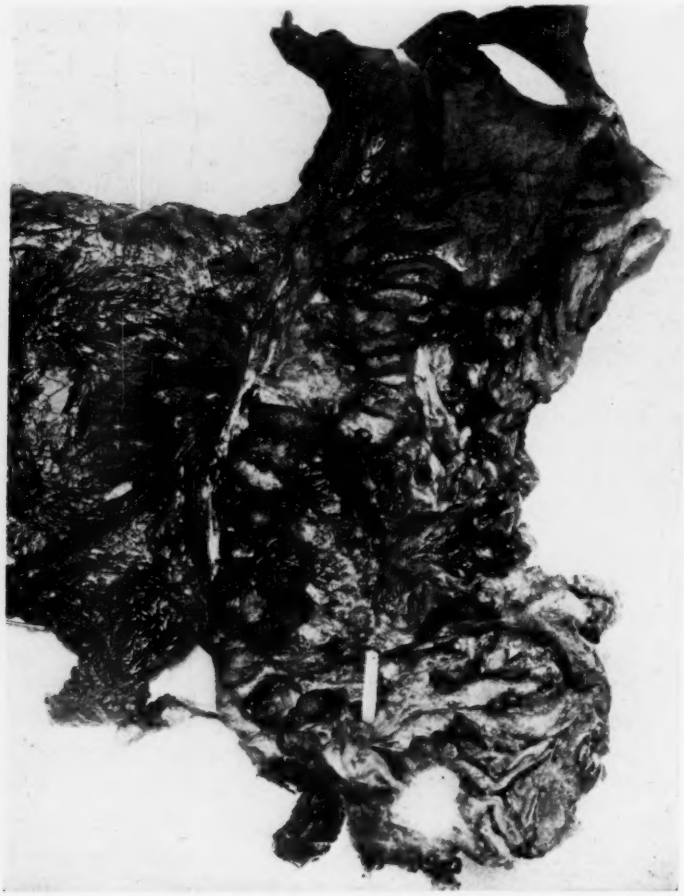

FIG. 5.—Hypertrophic colitis involving right colon. The extensive ulceration and polypoid hypertrophy of the mucosa are well shown. (Cf. Fig. 6.)

in every case either by abdominal palpation or by pelvic examination of the cul-de-sac. X-ray examination showed either an irregular filling defect or an area of narrowing in the involved segment. When the filling defect was unusually extensive, the presence of inflammatory disease rather than neoplastic disease seemed more likely. (Figs. 6, 7.) In two cases, radiological evidence of co-existing colitis in addition to the presence of a local lesion was shown. On the whole, however, radiological differentiation from

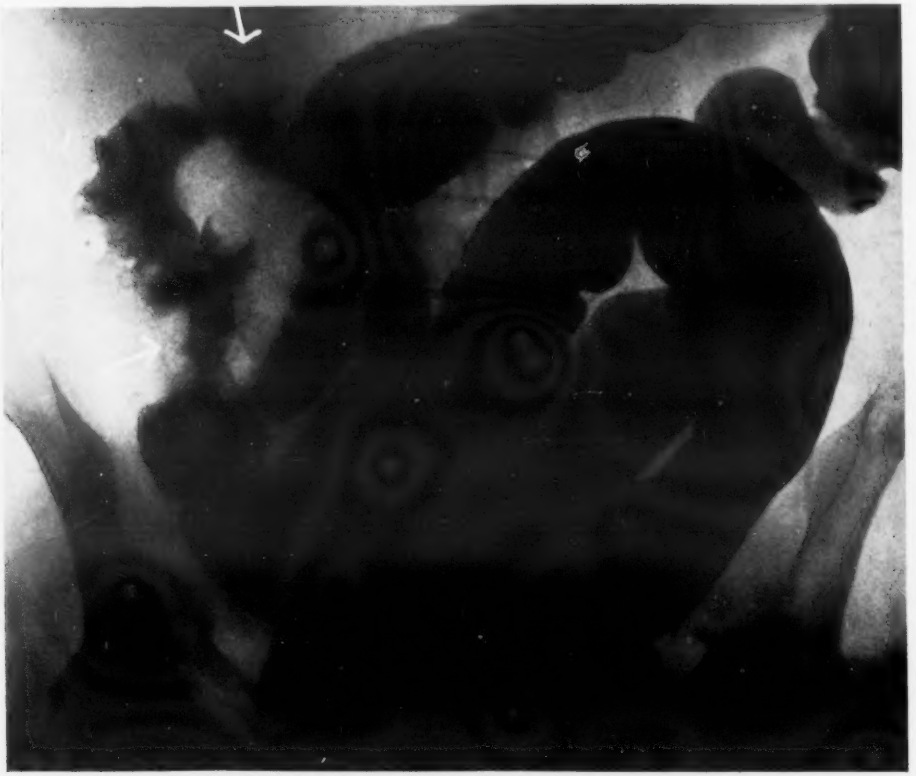

FIG. 6.—Case of localized hypertrophic ulcerative colitis—irregular filling defect involving ascending colon and hepatic flexure. The left half of transverse colon is normal.

FIG. 7.—Barium enema. Plate taken three years following resection, diseased area shown in Fig. 6. For two years following resection the transverse colon remained normal. Plate taken when patient had return of symptoms shows extension of diseased process to the transverse colon as far as the splenic flexure.

neoplasm or tuberculosis was not possible with certainty. The sigmoidoscope is of value in diagnosis not only for removing specimens for microscopical examination, but also for demonstrating multiple foci of disease.

At operation the determination of the exact nature of the pathological changes is again very difficult. Most of these cases were subjected to resection under the operative diagnosis of carcinoma or tuberculosis. Ulceration on the inside of the bowel, projection of papillary growths into the lumen and the presence of annular infiltration mimic neoplasm very closely.

In lesions involving the right side, ileocolic resection was performed five

FIG. 8.—Filling defect in left transverse colon caused by narrow annular structure, probably from healing of simple penetrating ulcer of colon.

times with no mortality; no other procedure was adopted for this form of the disease. One of the patients had a mild recurrence of symptoms which cleared up under dietetic treatment.

In lesions involving the upper and midsigmoid, a Mikulicz operation was performed three times and an exclusion ileosigmoidostomy in one case. In another case, where, in addition to a rectosigmoid mass, definite evidence of generalized colitis could be demonstrated, a cecostomy was performed. Marked shrinkage in size of the mass and subsidence in severity of the symptoms followed. In two cases with localized lesions at the rectosigmoid junction, abdominoperineal resection with restitution of continuity by extra-

peritoneal pre-sacral anastomosis was performed according to the technic of Dr. A. A. Berg. The only alternative in these cases would have been a left inguinal colostomy which would probably have resulted in complete stricture of the bowel below that site and precluded any later attempt at closure of the inguinal anus without a secondary and more difficult resection of the involved area. There were no mortalities in this group.

(V) SIMPLE PENETRATING ULCERS OF THE COLON

This term is applied to a group of cases which show one or more clean-cut penetrating ulcers which look almost like punched-out peptic ulcers. It is apparently a purely local disease, the surrounding colon not appearing to be grossly diseased. In two cases, penetration had occurred through the colonic wall and had become sealed off by adhesions of omentum or epiploic appendices with the formation of rather firm inflammatory masses which gave the impression of being penetrating or perforation neoplasms. Both these lesions were situated in the ascending colon and were operated upon with the clinical diagnosis of acute appendicitis. At operation they were mistaken for carcinoma of the colon and were resected as such. Another manifestation, probably the same type of lesion, was encountered at autopsy in a patient who had experienced severe repeated hæmorrhages from the bowel, one of which finally proved fatal. Twelve centimetres from the rectum a group of punched-out ulcers was encountered. At the base of one of them was an arteriosclerotic vessel which had been eroded by the penetrating ulcers.

The end-result of the healing of such an ulcer or group of ulcers was probably at the basis of an annular stricture following the transverse colon of a young woman with no evidences of colonic disease. We have no conception of the underlying etiology of these lesions. It is possible that they are due to injuries by ingested foreign bodies. It is also a possibility that they are of vascular origin and are due to the blocking of a small vessel. They have been found not infrequently at autopsies performed at this hospital in a large variety of conditions, especially uræmia, vascular diseases and blood dyscrasias. They are usually not matters of surgical concern except when by perforation they give rise to a mass or in the process of healing result in stricture.

(VI) INFLAMMATORY MASSES SECONDARY TO LESIONS OF APPENDAGES OF THE BOWEL (appendicitis, typhlitis, diverticulitis)

Probably the best known example of this variety of inflammatory mass is that which is secondary to *sigmoid diverticulitis*. This form has been so much discussed in recent years that we feel there is no reason for including it in the present study. We wish, however, to emphasize in passing that in addition to the large perisigmoidal inflammatory masses caused by perforation of a diverticulum or extension through it of infection from the lumen of the bowel, there is another and less common type. In the latter there is a gradually developing submucous inflammatory infiltration of the sigmoid as

well as an adhesive perisigmoiditis and the development of a considerable degree of intramural fibrosis and hyperplasia with a considerable degree of stenosis. This type clinically and radiologically is extremely difficult to differentiate from malignant stenosing lesions of the sigmoid and even at operation differentiation may be impossible.

The relation of the appendix to the development of certain hyperplastic masses in the ileocecal region is a moot point. In many cases an unresolved appendicitis is undoubtedly responsible for the formation of a hyperplastic fibrotic mass, the so-called *"appendicitis fibroplastica"* but this does not account for all the lesions found in this region. When the appendix is the source it is found buried in the cecal wall, or, as in one of our cases, in the terminal ileum, and forms part of the inflammatory mass. The extension of the inflammation in these cases is by contiguity, and, as might be expected, is mainly pericecal with involvement of the subserosal tissue. The submucous layer of the gut does not appear to be involved. Occasionally, tiny abscesses between the appendix and the cæcum will be uncovered when the former is mobilized, and fistulæ running into the cæcum may be found. This type of lesion is quite common, its true nature is usually appreciated and resection is rarely performed.

There is another and much rarer type of lesion, however, which may be called chronic typhlitis, in which the appendix, though thickened and indurated, lies free and non-adherent. Both appendix and cæcum show a marked submucous thickening, œdema and fibrosis. The lesion does not extend into the ascending colon or into the ileum except at the ileocecal valve, points which serve to differentiate it from the two other types of non-specific inflammatory disease encountered in this region which have been discussed above. Large masses of firm nodes are found in the ileocecal angle. Upon examination the pathological alterations involving the appendix and cæcum are seen to be continuous. If the route of spread were by direct extension from appendix into cæcum it would have to be through the contiguous submucous layers of the appendix and cæcum. However, clinically it is well known that inflammation of the appendix usually stops short of the extreme base even in the most virulent form of the disease. Doctor Klemperer, who at one time made a study of the extent of the basal involvement in acute appendicitis, was able to substantiate this clinical observation from his pathological study.

The question then arises whether the extension takes place into the cæcum from some unusual form of acute appendicitis involving the base or whether the ileocecal changes are primary, the appendix participating simply as a component portion of this segment of gut. We are inclined to believe that the type of chronic cecitis which shows extensive submucous intramural involvement but without evidences of mucosal ulceration is secondary to a partially resolved acute or chronic typhlitis.

There is no doubt about the existence of acute typhlitis as a clinical entity. On an active emergency service three or four such cases are encountered

every year. Clinically and on physical examination they present the picture of acute appendicitis. Operation, however, reveals a succulent, œdematous, inflammatory lesion without much peritoneal injection or fibrin deposition, involving the cæcum, the retroperitoneal tissue, the appendix and the ileocecal glands. The appendix does not appear to be more acutely involved than any of the adjacent tissues. Localized areas of induration from ½ inch to an inch in diameter may be present. In some of these cases the appendix and a lymph-node from the ileocecal angle were removed. On pathological examination these revealed only acute inflammatory hyperplasia. In one subacute case in which ileocecal resection was performed, a small ulcer was still present in the cæcum; the submucosal proliferative reaction was out of all proportion to the size of the ulcerative lesion. Most of the acute cases probably clear up, with or without operation. An especially severe case was recently encountered which came to post-mortem examination. The cæcum was found greenish-black and gangrenous. There were numerous cecal ulcerations, two of which had perforated. The appendix was gangrenous. Jennings[8] has recently called attention to this type of case.

In other cases repeated attacks result finally in the formation of a chronic submucous and subserous inflammatory infiltration. In such chronic cases there are no ulcerative, polypoid or papillary changes in the mucosa; the cecal wall is thickened and indurated and there are few adhesions present. A chronic lymphadenitis of the ileocecal lymph-nodes may contribute to the final pathological picture.

The symptom usually complained of is recurrent pain in the right lower quadrant without any history of blood in the stool, diarrhœa or constipation. At times the chief complaint is the presence of a mass. Radiologically, filling defects or irregularities in the cæcum are noted. The general condition of the patient is usually good, operation being mainly undertaken because of the presence of a mass. At operation differentiation from tuberculosis may be difficult and the cases subjected to resection have been operated on because of their similarity to that condition.

SUMMARY

(1) A study is reported of fifty-two cases, exclusive of sigmoid diverticulitis, manifesting themselves clinically as tumors or strictures of the bowel.

(2) Clinically, radiologically and at operation these were usually regarded as malignancy or localized hyperplastic tuberculosis.

(3) Microscopical examination of the resected specimens showed various stages and degrees of acute and chronic inflammation with production of much fibrous tissue.

(4) No exact pathological or etiological classification is attempted. For clinical purposes the cases are divided into six groups, some of which overlap:

(*a*) Pericolonic or peri-intestinal granulomata due to sealed-off perforations.

(*b*) Intestinal stenosis due to known vascular lesions of the bowel.

(*c*) Localized hypertrophic ulcerative ileitis.

(*d*) Localized hypertrophic colitis.

(*e*) Local penetrating ulcers of the colon.

(*f*) Granulomata secondary to inflammation of appendages or diverticula of the bowel.

(5) The various groups are discussed and illustrative cases briefly reported.

(6) In our material localized hyperplastic tuberculosis of the bowel in patients without evidence of pulmonary tuberculosis was less common than the non-specific variety of inflammatory lesion of the bowel.

The authors wish to extend their gratitude to Dr. A. A. Berg for so generously placing his extensive material at their disposal. To Dr. Paul Klemperer they are indebted for his invaluable and gracious assistance in studying the pathological phases of the conditions considered in this paper. Thanks are due to Dr. Louis Gross for numerous suggestions in the preparation of this paper and for putting the facilities of the laboratory at our disposal. To Drs. Richard Lewisohn, Harold Neuhof, Edwin Beer and I. C. Rubin thanks are due for permission to use individual cases.

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ENTEROSTOMY IN ILEUS

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The prognosis of acute intestinal obstruction has always been grave. In spite of the numerous clinical and important experimental studies made in this field, little of practical value has been added to lessen the staggering mortality.

The actual cause and mechanism of death in these cases is obscure and the fact that so much controversy exists emphasizes quite definitely that the problem is far from its ultimate solution. It is not within the province of the present study to discuss this phase of intestinal obstruction, for the recent papers of Ochsner[1] and Morton[2] have fully covered this material. It may suffice to state that the majority of investigators believe that toxins are produced in the obstructed bowel, which, when absorbed, cause the familiar clinical picture, but the exact nature, origin, composition and manner of absorption of these poisons is still undetermined. There is no doubt that cases of duodenal and jejunal obstruction run a more rapid and fatal course than those in which either the ileum or colon are affected. The loss of the secretions of the upper portion of the gastro-intestinal tract probably accounts for this. It is possible that the toxæmia in high obstructions may be increased by the absorption of duodenal and pancreatic secretions before they can be adequately detoxified by the cells of the ileum. It is also true that cases of obstruction complicated by a vascular strangulation are more toxic than those in which the obstruction is simple. But, undoubtedly, the most important factor influencing the prognosis is the element of time, for naturally the longer the period of obstruction, the greater the absorption of intestinal toxins, and the more marked the dehydration. Most workers are agreed that the characteristic dehydration and the concomitant chemical changes in the blood, the increase in the urea and non-protein nitrogen, the decrease in the plasma chlorides, and the rise in the carbon dioxide combining power, play very important rôles in the fatal cases of intestinal obstruction.

The clinical picture of either the mechanical or the paralytic variety of intestinal obstruction is quite similar and is usually evidenced by severe dehydration, abdominal distention with or without vomiting, the absence of bowel evacuations, and occasionally visible peristalsis. It is very important, however, to differentiate between these two varieties because the treatment is different.

The main basis of therapy in dynamic ileus must be the urgent relief of the obstruction with its toxæmia and the associated dehydration. An early diagnosis with immediate operation and the adequate supply of fluids administered orally, subcutaneously, intravenously and by rectum, answer these requirements.

Whenever possible, the anæsthesia employed should be spinal because it insures a complete abdominal relaxation which makes exposure simple, and the operation may be speedily performed with the minimum amount of trauma and shock. There is a group of cases in which the obstructed intestine remains dilated after the cause has been removed. The question naturally arises whether the immediate external drainage of the distended loops will diminish the degree of intestinal toxæmia and relieve intra-intestinal pressure. The operation of enterostomy has been used for years. When the shortcomings of ileostomy were recognized more than a decade ago, jejunostomy was enthusiastically recommended. High jejunostomy for a time was heralded in many clinics as an ideal method to effectively drain the lethal toxins originating in the duodenum. Theoretically, it should lessen the absorptions of intestinal poisons near the source of their origin. The arguments in favor of jejunostomy, however, seem to be mainly academic for any surgeon who has seen the rapid deterioration of unfortunate patients suffering from duodenal fistulæ, has little enthusiasm for the possible problem of a subsequent persistent high jejunal fistula. For even though fluids are adequately supplied to combat the dehydration caused by the loss of bile, duodenal and pancreatic secretions, death often occurs. In fact Haden and Orr[3] who have contributed much of value to the vast bibliography of intestinal obstruction have shown that in experimental high obstruction in dogs, a jejunostomy shortened the span of life. And while ileostomy itself did not prolong the lives of the animals, if this procedure was combined with hypodermocylsis, life was definitely prolonged.

Cases have been observed clinically in which a jejunostomy was performed to insure the immediate drainage of high intestinal toxins, but the life of the individual became so seriously menaced by subsequent dehydration, inanition and skin maceration, that the closure of the jejunal fistula was demanded.

Ileostomy in general and jejunostomy in particular have never been used extensively in the treatment of intestinal obstruction at Mount Sinai Hospital, New York. A study of the available material will show that jejunostomy has not demonstrated its superiority sufficiently to counterbalance the ill-results seen so often after its institution, and even ileostomy has been of such doubtful value that it should only be used in very carefully selected cases.

In a large series of cases of intestinal obstruction of various groups, enterostomy was done in only forty-one cases of the acute mechanical ileus and in twenty-one cases of paralytic obstruction. The causes of the ileus for which an enterostomy was performed are enumerated in Tables I and II respectively.

The mortality following enterostomy in the dynamic group averaged about 70 per cent., and in the adynamic, 80 per cent. Jejunostomy was done in eleven cases with a mortality of 73 per cent., and ileostomy in the remainder with a mortality of 74 per cent. This is a fearful mortality but it must not be forgotten that in the majority of cases, the period of obstruction had lasted from one to six days, and in many instances, enterostomy was done as a pro-

cedure of last resort and performed in a desperate endeavor to stave off impending dissolution.

TABLE I

Cases of Mechanical Ileus in Which Enterostomy Was Performed

Cause of Obstruction	Number	Living	Dead	Percentage Mortality
Post-operative adhesions old	15	3	12	
Tumors of the large bowel	8	3	5	
Acute intussusception	7	0	7	
Strangulated hernia	4	0	4	
Post-operative adhesions following recent appendicectomy	4	4	0	
Inflammatory ileal stricture	2	1	1	
Pelvic peritonitis following carcinoma of bladder	1	0	1	
Pelvic peritonitis following diverticulum of bladder	1	1	0	
Pelvic peritonitis following appendicectomy	2	2	0	
	44	14	30	70%

The Witzel technic of enterostomy was performed in almost all cases. While it is a simple operation, it requires great care and careful attention to the toilet of the peritoneum. The insertion of a rubber tube into a friable, dilated intestine filled with highly toxic material, may result in the tearing of the bowel with inevitable spilling and a generalized contamination unless the procedure is gently performed. The cuff of the enterostomy tube must be securely fixed to the surrounding skin for otherwise the tube may be inadvertently dislodged causing intraperitoneal leakage. This unfortunate accident happened twice in this series. In another case the indwelling tube caused pressure against the bowel wall resulting in a perforation into the free peritoneal cavity. After a certain length of time, leakage occurs about the tube and the care of the surrounding skin taxes the ingenuity of most resourceful surgeons. The automatic closure of the enterostomy following the withdrawal of the tube does not happen as frequently as a theoretical consideration of the Witzel valve might lead one to believe. The enteric fistula in the sixteen cases which survived did not close immediately or spontaneously upon the withdrawal of the tube. The enterostomy often took weeks to close, and in eight operations for closure were necessary at intervals from fourteen to eighty days following intestinal drainage. The operative closure of any fistula is not a simple problem. It is not always successful and in two cases a recurrence took place. Death resulted in another from peritonitis due to a noncompetent anastomosis following an enterorrhaphy.

It might be advisable at this point to turn from a general discussion to a consideration of the value of enterostomy in certain selected groups of cases in which it was instituted.

The first group of cases which illustrates the value of enterostomy in a mechanical type of obstruction consists of 135 cases of acute ileus due to old post-operative adhesions. The operative procedure in the majority of 120 cases was simple division of the obstructing band although in a few, an additional entero-enterostomy was performed.

Twenty-four of this number died, a mortality of about 20 per cent. Enterostomy was added as an additional procedure in fifteen cases, or 11 per cent. of the total number. The average period of obstruction in these cases was eighty hours. The mortality in this enterostomized group was 80 per cent.

Enterostomy was performed in twelve at the time of the primary operation after the obstructing band had been removed, and in three as a secondary procedure after the signs of obstruction still persisted after the first operation. Eight succumbed in the primary group, four from the effects of the paralytic ileus, two from peritonitis, one from shock, and one from pneumonia. All died in the secondary group from a persistent paralytic ileus. In four of the eleven patients who died, the intestinal drainage from the enterostomy tube was charted as satisfactory. But in spite of this death resulted. These patients had probably absorbed the lethal dose of intestinal toxin prior to the first operation or subsequent to the second, and, therefore, were marked for death regardless of any surgical interference.

TABLE II

Cases of Paralytic Ileus in Which Enterostomy Was Performed

Type	Number	Living	Dead	Percentage Mortality
Acute appendicitis with general peritonitis	9	2	7	
Perforated gastroduodenal ulcer with general peritonitis	4	0	4	
Perforated gall-bladder with bile peritonitis	2	0	2	
Perforated typhoid ulcer with general peritonitis	2	1	1	
Perforated carcinoma of stomach with general peritonitis	1	0	1	
Perforated diverticulum of sigmoid with general peritonitis	1	0	1	
Perforated Meckel's diverticulum with general peritonitis	1	0	1	
Mesenteric thrombosis	1	0	1	
	21	3	18	85%

The second group of cases illustrative of the value of enterostomy in mechanical obstruction consists of 278 strangulated hernias of various kinds, many of which were associated with vascular compromise. The obstruction was relieved at a very early period in 199. These were relatively non-toxic cases in which the obstruction was simple and without vascular strangulation. The mortality of this group was 13 per cent. But the mortality climbed to

29 per cent. in forty-seven cases in which operation was deferred until the obstructed bowel exhibited those characteristic changes dependent upon vascular strangulation. The mortality arose to 40 per cent. in twenty-two cases in which a frank gangrene of the obstructed intestinal loop necessitated resection. The high death rates in these two aforementioned groups were undoubtedly attributable to the more severe toxæmia which invariably accompanies any obstruction complicated by vascular strangulation. It is in this type of severely toxic case that enterostomy should be indicated. For if the immediate drainage of intestinal toxins is of any consequence, it should be evidenced here. Immediate drainage of the highly toxic intestinal content should tide the patient over the critical period until peristalsis has been normally resumed. Enterostomy was done in four cases and all these terminated fatally. The number is small and it is difficult to draw any conclusions. But it does seem logical to conclude that the cases operated on early are those of simple obstruction in which the minimal amount of intestinal toxin, if any, has been absorbed. Therefore, the degree of intestinal paresis is slight and the muscular tone of the intestinal wall is good. Normal peristalsis is soon resumed with a free evacuation of the bowel. On the other hand, cases of intestinal obstruction operated late are those complicated by vascular strangulation with its incident and concomitant changes. Larger quantities and more toxic secretions and excretions are absorbed from the dilated necrotized intestine. Intestinal paresis is marked, muscular tone is lacking, and the peristalsis is practically nil. This paralytic condition may persist for a varying period after the obstruction has been relieved. What reason is there to believe that a paralyzed bowel will drain better if an artificial drainage tract is established? It will drain only locally, and, therefore, does little to relieve the general intraintestinal pressure and toxæmia. The fistula may drain later when intestinal peristalsis returns, but at that time the lethal dose of intestinal toxins may have been absorbed, or peristalsis has returned to such a degree that enemata have become effectual. There is little need for an enterostomy at this time. One group of cases of intestinal obstruction definitely benefited by enterostomy were those caused by tumors of the large bowel. While many surgeons prefer a cæcostomy in these cases, a low ileostomy offered immediate relief from the obstruction in three of the seven cases in which a preliminary emergency drainage procedure was done. When the condition of the patients had improved sufficiently, the more radical procedure of colonic resection was subsequently performed.

It might be instructive to review the frequency of enterostomy in cases of acute appendicitis. Intestinal obstruction may occur soon after appendicectomy due either to recent intraperitoneal adhesions or bands, or pelvic exudate, or the result of paralytic ileus. Two thousand, eight hundred and forty-one cases were operated on during a ten-year period. The type of appendicitis and the frequency of enterostomy for intestinal obstruction is given in Table III.

TABLE III

Type	No.	Mortality	Enterostomy as a Secondary Procedure	Died After Enterostomy
Catarrhal appendicitis	82	.95	0	0
Acute gangrenous appendicitis	973	3.1	2	1
Acute appendicitis with abscess	665	5.4	2	0
Acute appendicitis with generalized peritonitis	359	20.	11	6

Enterostomy was performed in fifteen cases of acute appendicitis. Six were performed because of a secondary mechanical obstruction occurring soon after appendicectomy. Two of these were caused by pelvic exudation in which jejunostomy alone was done, and four were caused by recent adhesions in which obstructing bands were divided and an ileostomy performed. All six cases recovered. The remaining nine cases for which enterostomy was instituted were due to a paralytic ileus; only two of these patients recovered.

Enterostomy in the paralytic variety of intestinal obstruction has been practically discarded. Its usefulness can best be evaluated reviewing the twenty-one cases enumerated in Table II in which an enterostomy was performed for paralytic ileus incident to a general peritonitis. The mortality was 85 per cent.

Most surgeons are agreed that external intestinal drainage accomplishes little when the peritoneum is diffusely inflamed, and the intestines bound together and kinked by adhesions are paralyzed and atonic. The dry enterostomy tube is the usual proof of the inadequacy of this procedure.

Enterostomy apparently has no indication in the treatment of adynamic ileus. The use of irritative enemas, turpentine stupes, the judicious administration of pituitrin, the maintenance of a water balance, and the use of the Levin tube are certainly more efficacious than external intestinal drainage. Satisfactory retrograde drainage of duodenal, pancreatic and biliary secretions has been accomplished without the aid of an external jejunal or ileal fistula by the intranasal introduction of the Levin gastric tube. When once inserted, it is usually well tolerated by the patient and may be left in the stomach for days. In this respect alone, it is far superior to the intermittent gastric lavage which taxes the strength of the debilitated patient. The Levin tube acts as an effective drain for the gastric and the refluxed intestinal contents, and acts prophylactically against a gastric dilatation. If drainage is sluggish, it can be aided by aspiration through the tube. The danger of an alkalosis from the loss of gastric secretions can be guarded against by the liberal intravenous administration of saline. In addition, the thirst of patients is psychically relieved for they may drink liberally inasmuch as the ingested fluid is automatically withdrawn by syphonage. When the clinical condition of the patient improves, as may be determined by the character and amount of gastric return, the diminution of abdominal distention, and the satisfactory return

from enemata, the Levin tube may be intermittently clamped. It may be withdrawn as soon as the normal intestinal flow is established.

SUMMARY AND CONCLUSIONS.—It would appear from this study of forty-four cases of acute mechanical ileus in which an enterostomy was done, that it was of benefit in a selected group of cases only.

One group consisted of those cases of obstruction due to localized inflammatory adhesions seen in pelvic exudates. The local infection would have been widely spread if an operative attempt had been made to free the obstruction at the site of origin. An enterostomy in these cases is logical as a makeshift procedure and can usually be performed in a fairly clean field above the point of obstruction. It will usually tide the patient over the acute stage of an obstruction until the infection has subsided sufficiently to allow a more thorough exploration subsequently, and occasionally it will be all that is necessary. Clinical experience will testify to its efficacy as an emergency procedure. This was successfully done in three cases of obstruction incident to pelvic peritonitis. As the infection subsided and the obstruction was automatically relieved, the normal intestinal passage was restored and the enterostomy closed spontaneously.

The other group of cases apparently aided by enterostomy are those due to obstructive tumors of the colon. While many prefer a cæcostomy, an ileostomy may offer temporary relief from the obstructive symptoms until the condition of the patient has improved sufficiently to permit subsequent colonic resections.

It is extremely doubtful, however, whether enterostomy is of benefit in other groups of cases in which the cause of obstruction has been relieved. Ileostomy as an additional procedure in relieving intestinal toxæmia has little to offer and jejunostomy less. In many of those cases which recovered, enterostomy seems to have been an added source of danger because of a persistently draining enteric fistula. In fact, at present, the high mortality of intestinal obstruction of mechanical origin can only hope to be lowered if the condition is diagnosed early. The cause of the obstruction must be actively relieved by immediate operation under spinal anæsthesia. The dehydration present should always be adequately relieved by the liberal administration of fluid.

Enterostomy has no indication in the paralytic type of ileus.

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INTUSSUSCEPTION DUE TO INVAGINATED MECKEL'S DIVERTICULUM

REPORT OF TWO CASES WITH A STUDY OF 160 CASES COLLECTED FROM THE LITERATURE

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Meckel's diverticulum and intussusception are relatively common, but the combination is quite infrequent. Reports of two cases of this combination operated upon at the University of Chicago clinics in the past year are included in the present article. An attempt is made to formulate a clinical syndrome from an analysis of these two cases and from those reported in the literature.

Report of Cases.—Case LXXII.*—U. M., male infant, aged seven months, entered the hospital with bloody stools, vomiting, abdominal pain, and signs of intestinal obstruction of three days' duration. The patient was the first child in the family and his birth and feeding history were normal. He was entirely well and had no signs of abdominal distress except three weeks before the present illness. At that time he passed two bloody stools and was indefinitely ailing for a period of twenty-four hours, but recovered completely.

The present illness began suddenly with vomiting and bloody stools. These symptoms disappeared after the patient had received an enema. He seemed almost entirely well until about twelve hours before entrance when he again had vomiting and bloody stools and was apparently suffering great pain. There was no evidence that the pain was of a colicky type. The abdomen was markedly distended, there was no tumor palpable on either rectal or abdominal examination, there could be no certainty as to whether there was tenderness because of the child's age, and there was no rigidity. The temperature was 37.0° C., and the white blood count, 11,000. During fluoroscopic examination of the colon, the barium passed unhindered up to the splenic flexure where it stopped suddenly with the typical bulbous expansion with a cup-like depression in its centre suggestive of intussusception.

Operation was performed by Doctor Andrews three days after the onset and two hours after entrance to the hospital. A combination of novocaine locally and ethylene-ether as a general anæsthetic was used. No excessive free fluid was found in the peritoneal cavity. An intussusception was found with four inches of ileum prolapsed through the ileocæcal valve. This was reduced by expulsion and at the apex of the intussusceptum was found a Meckel's diverticulum about one centimetre in length. By squeezing the intestine, the diverticulum was everted and was so œdematous that it stayed everted. There was no gangrene and no further operative procedure was done. There was no tumor of the diverticulum. The abdomen was closed without drainage. The child is now well, eleven months after operation.

Case LXXIII.—W. P., male, aged five, entered the hospital with vomiting, colicky pains around the umbilicus, and absolute constipation of thirty-two hours' duration. The

* The numbers of this and of the following case correspond with those given in a tabular summary later in the article. Case LXXII has been previously reported by Doctor Andrews.

patient was the third of several children and his birth and feeding history were normal. He was always well and there was no history of previous abdominal attacks.

The present illness began at 8 A.M. on January 5, 1932, with sudden onset of severe, cramp-like pain all over the abdomen, but somewhat localized to the region of the umbilicus. Some blood was returned from an enema, there was vomiting, and the white blood count was 16,500. The patient was given paregoric and slept well that night. The morning of January 6 the pains returned with increased intensity at five- to ten-minute intervals. Between the paroxysms there was comparative comfort. The colicky nature of the pains and their localization to the region of the umbilicus was more definite than on the preceding day. At 3 P.M. the patient was admitted to the hospital. No mass could be felt by abdominal or rectal examination. There was tenderness all over the abdomen, most marked in the right lower quadrant. There was no distention and no rigidity. The temperature was 37.5° C.; pulse rate, 118; hæmoglobin 85 per cent.; red cells, 4,800,000; and white cells, 13,500 with 60 per cent. polymorphonuclears and 40 per cent. mononuclears. The urine was normal.

Operation was performed by Doctor Andrews at 4:00 P.M., thirty-two hours after the onset and one hour after admission to the hospital. The anæsthetic was ethylene and ether. A right rectus incision was made and when the peritoneal cavity was opened, considerable serous fluid escaped. An ileocæcal intussusception about twenty-five centimetres long was found and easily reduced by expulsion and milking. At the apex of the intussusception was an inverted Meckel's diverticulum about four centimetres long. This was everted by squeezing the bowel and was gangrenous. The diverticulum was excised and the longitudinal opening on the antimesenteric surface of the bowel closed. The abdomen was closed without drainage. For three days the patient vomited considerably. Glucose in Ringer's solution and glucose alone were given subcutaneously and rectally. There was a spontaneous stool and gas on the third day. There was no vomiting after January 9, a dry diet being started on this day, and thereafter recovery was uneventful. The diverticulum showed no evidence of tumor and histological examination showed necrosis, especially of the mucous layer. The child is now well, three months after operation.

SUMMARY OF LITERATURE.—The earliest known case of intussusception due to invaginated Meckel's diverticulum is the specimen placed in the St. Bartholomew's Hospital Anatomical Museum in 1846. I am able to find 160 cases recorded in the literature. Cheyne[19] reviewed sixteen cases in 1904; Forgue and Riche,[42] thirty-four in 1907; Gray,[49] forty in 1908; Gross,[51] 40 in 1912; Kasemeyer,[72] forty-two in 1912; Hertzler and Gibson,[61] forty-five in 1913; Wellington,[134] fifty-nine in 1913; Lower,[86] fifty-two in 1925; and Kaspar,[74] seventy-two cases in 1925.

The cases reviewed in the present article are divided into two groups. Group I includes 114 cases with relatively complete individual case reports. On the basis of Group I, an analysis is made of the symptoms, course and treatment of intussusception due to invaginated Meckel's diverticulum. Group II includes forty-six cases with incomplete individual case reports and in the analysis of the literature this group is not considered so as not to distort the averages and percentages.

REPORTED CASES: GROUP I

Series of 114 cases of intussusception due to invaginated Meckel's diverticulum for which enough data are given to make them the basis of an analysis later in this article. In all the 114 cases, the following facts are tabulated: case number, author, date, age of patient in years, and all other factors listed in the following summation of the group. In

all instances where any of these facts are not mentioned in the case abstracts, data were not given concerning them in the original reports.

Age.—Average in 113 cases with data given on this point, thirteen years.

Sex.—In 105 cases with data given, eighty-three males and twenty-two females.

Previous abdominal crises were present in forty-six out of sixty-seven cases with data given.

Abdominal pain was present in ninety-seven out of ninety-eight cases with data given.

Vomiting was present in eighty-two out of eighty-two cases with data given.

Meteorism was present in forty-five out of sixty-nine cases with data given.

Abdominal tenderness was present in forty-one out of fifty-four cases with data given.

Abdominal rigidity was present in twenty out of forty-two cases with data given.

Mass palpable per rectum was present in four out of thirty-seven cases with data given.

Tumor in right lower quadrant was present in forty-one out of eighty-one cases with data given.

Colic was present in fifty-two out of fifty-four cases with data given.

Fever over 37.5° C. was present in fifteen out of thirty-nine cases with data given.

Blood was passed per rectum in forty-three out of seventy-seven cases with data given.

Average duration of the present illness was 81.6 hours in eighty-eight cases with data given.

Abdominal exudate was found in thirty-nine out of forty-three cases with data given.

Diverticulum contained a tumor in twenty-six out of sixty-three cases with data given.

Gangrene was present in thirty-nine out of seventy-one cases with data given.

Diverticulum was reducible in fifty-four out of eighty-nine cases with data given.

Resection of the diverticulum only was performed in thirty-nine out of 113 cases with data given.

Resection of the bowel was performed in sixty-one out of 114 cases with data given.

Recovery occurred in sixty-six out of 112 cases with data given.

CASE I.—Ingle,[66] 1888, age five months, pain, vomiting, meteorism, no tenderness, no tumor in right lower quadrant, melena, duration, five days; operation not permitted; death.

CASE II.—Weil and Frankel,[133] 1896, age four years, female, no previous attacks; present attack: pain, vomiting, no meteorism, tenderness, no rigidity, tumor in right lower quadrant, temperature 37° C., melena, duration, thirty-six hours; operation: abdominal exudate, diverticulum had no tumor, was gangrenous and was reducible, resection of bowel; death.

CASE III.—Ewald,[38] 1897, age forty-two years, female, several attacks of abdominal pain in past seven months; present attack: pain of colicky type, vomiting, meteorism, no melena, diverticulum contained no tumor but was gangrenous, no operation; death.

CASE IV.—Küttner,[82] 1898, age forty-nine years, female, acute attack eight months before; present attack: pain, vomiting, meteorism, no rigidity, no tumor in right lower quadrant, no mass felt per rectum, fever 39.2° C., no melena, duration, three days; operation: abdominal exudate, diverticulum had no tumor, was gangrenous and not reducible, intestinal anastomosis only, no reduction of intussusception; death.

CASE V.—Haasler,[56] 1902, age thirty-five years, male, attack of abdominal pain one year before; present attack: pain of colicky type, vomiting, meteorism, no tenderness, no rigidity, no tumor in right lower quadrant, no mass felt per rectum, no melena, duration, seven days; operation: abdominal exudate, diverticulum contained a tumor, was reducible, but not gangrenous, resection of bowel with colostomy and no anastomosis; death.

CASE VI.—Bize,[8] 1904, aged six years, female, no previous distress; present attack: pain, vomiting, meteorism, tenderness, no tumor in right lower quadrant, fever 38° C., melena, duration, five days; operation: abdominal exudate, diverticulum contained a tumor, was reducible, but not gangrenous, resection of diverticulum only; death.

CASE VII.—Bousquet,[10] 1904, age thirty-nine years, male, bloody stools with colic for four months, abdominal mass that disappeared two weeks before onset; present attack: colicky pain; vomiting, meteorism, no tenderness, no rigidity, tumor in right lower quadrant, no fever, duration, two days; operation: abdominal exudate, diverticulum contained a tumor, was not gangrenous, but was reducible, resection of diverticulum only; recovery.

CASE VIII.—Rehn,[112a] 1904, aged thirty years, male, attacks of abdominal pain for months; present attack: colicky pain, tumor in right lower quadrant, operation: tumor of diverticulum, resection of bowel; recovery.

CASE IX.—Hirschsprung,[63] 1905, age three years, male, melena for three days, resection of bowel; death.

CASE X.—Richter,[113] 1906, age three years, male, no previous abdominal complaint except obstipation; present attack: pain, vomiting, meteorism, no tumor in right lower quadrant, no mass felt per rectum, temperature 37.6° C., melena, duration, one day; operation: diverticulum reducible, resection of diverticulum only; death.

CASE XI.—Mothersole,[101] 1907, age six years, male, present attack: pain, vomiting, tenderness, rigidity, no tumor in right lower quadrant, melena, duration, one day; operation: diverticulum contained no tumor, was reducible and not gangrenous, only the diverticulum was invaginated, the ileum not forming part of the intussusceptum, resection of the diverticulum only; recovery.

CASE XII.—Gruson,[52] 1907, age twenty-five years, male, previous abdominal distress; present attack: colicky pain, vomiting, meteorism, tenderness, rigidity, no tumor in right lower quadrant, no mass felt per rectum, temperature 35° C., no melena, duration fourteen hours, abdominal exudate, diverticulum contained a tumor, but was not gangrenous, no operation; death.

CASE XIII.—Haeberlin,[57] 1908, age three years, female, melena at birth, vague gastroenteritis for one year; present illness: colicky pain, vomiting, no meteorism, tenderness, rigidity, tumor in right lower quadrant, no melena, duration, fourteen days of daily cramps; operation: diverticulum contained no tumor, was not gangrenous and was reducible, resection of diverticulum only by a wedge-shaped incision; recovery.

CASE XIV.—Gaudier,[47] 1909, age twelve years, male; present illness: colicky pain with visible peristaltic waves, vomiting, no meteorism, no melena, duration four days; operation: abdominal exudate, diverticulum contained no tumor and was not reducible, resection of bowel with end-to-end anastomosis; recovery.

CASE XV.—Moore,[98] 1911, age twenty-five years, attacks of colic for several years; present illness: colicky pain, duration, one day; diverticulum contained no tumor, was not gangrenous and was reducible; operation: resection of diverticulum only; recovery.

CASE XVI.—Osmanski,[106] 1911, age seven years, female; present illness: pain, vomiting, meteorism, tenderness, no tumor in right lower quadrant, no melena, duration two days; operation: abdominal exudate, diverticulum contained a tumor, was not gangrenous and was reducible, resection of diverticulum only; recovery.

CASE XVII.—Osmanski,[106] 1911, age eleven years, male; present illness: colicky pain, vomiting, meteorism, extreme tenderness, no tumor in right lower quadrant, no melena, duration, three days; operation: abdominal exudate, diverticulum reducible, resection of diverticulum only, puncture of a distended loop of bowel with a trocar; recovery.

CASE XVIII.—Gulecke,[54] 1912, age five years; present illness: colicky pain, tenderness, tumor in right lower quadrant, no mass felt per rectum, bloody diarrhœa, duration, fourteen days; operation: diverticulum contained a tumor and was reducible, resection of diverticulum only; recovery.

CASE XIX.—Gaardlund,[46] 1912, age fourteen years, male, abdominal pain seven years before; present illness: colicky pain, vomiting, no meteorism, tenderness, no rigidity, tumor in right lower quadrant, mass felt per rectum, temperature 37.5° C., melena, duration, nine hours; operation: no abdominal exudate, diverticulum contained a tumor,

was gangrenous and was reducible, resection of diverticulum only, subsequent bowel resection; recovery.

CASE XX.—Höpfner,[67] 1912, age nine years, male, no previous attacks; present illness: pain, vomiting, meteorism, tenderness, rigidity, no tumor in right lower quadrant, no mass felt per rectum, no fever, duration, one day; operation: abdominal exudate, diverticulum contained a tumor, was gangrenous, and was not reducible, resection of bowel; recovery.

CASE XXI.—Marquis,[80] 1913, age three years; present illness: pain, tumor in right lower quadrant, melena, duration, thirty-six hours; operation: diverticulum contained no tumor, was not gangrenous and was reducible, resection of diverticulum only; recovery.

CASE XXII.—Brennecke,[12] 1913, age five years, male, previous abdominal distress; present illness: pain, vomiting, melena, duration, one day; operation: diverticulum contained no tumor, was gangrenous and not reducible, resection of bowel; recovery.

CASE XXIII.—Kaspar,[73] 1914, age thirteen years, male; present illness: colicky pain, fæcal vomiting, meteorism, tenderness, no tumor in right lower quadrant, temperature 37.6° C., duration, twelve days; operation: abdominal exudate, diverticulum contained a tumor, was gangrenous and was reducible, resection of diverticulum only and side-to-side anastomosis of small bowel; death.

CASE XXIV.—Fromme,[44] 1914, age seven years, male, no previous abdominal distress; present illness: pain, vomiting, tenderness, rigidity, tumor in right lower quadrant, temperature 37.5° C., melena, duration, two days; operation: diverticulum contained no tumor, was gangrenous and was reducible, resection of bowel; recovery.

CASE XXV.—Fauntleroy,[40] 1916, age twenty-four years, male, no previous abdominal distress; present illness: colicky pain, vomiting, meteorism, no tumor in right lower quadrant, no mass felt per rectum, temperature 36.6° C., no melena, duration, two hours; operation: diverticulum contained no tumor, was not gangrenous and was reducible, the bowel was gangrenous, resection of diverticulum and resection of bowel because diverticulum was not on the gangrenous portion of the bowel; recovery.

CASE XXVI.—Vangsted,[129] 1918, age ten years, female; present illness: pain of two days' duration; operation: abdominal exudate, resection of bowel; recovery.

CASE XXVII.—Carlson,[17] 1919, age eighteen years, male, had abdominal pain day before onset; present illness: colicky pain, vomiting, no meteorism, tenderness, no rigidity, no tumor in right lower quadrant, no mass felt per rectum, no fever, no melena, duration, two days; operation: abdominal exudate, diverticulum contained no tumor, was not gangrenous and was reducible, resection of diverticulum only; recovery.

CASE XXVIII.—Carlson,[17] 1919, age four years, male, often complained of griping pains, abdominal pain two days before onset; present illness: colicky pain, vomiting, no meteorism, tenderness, no rigidity, tumor in right lower quadrant, no mass felt per rectum, no melena, duration, seven hours; operation: diverticulum contained no tumor, was not gangrenous and was not reducible, resection of bowel; recovery.

CASE XXIX.—Guibé,[53] 1919, age four years; previous attack five days before with complete recovery; present illness: pain, vomiting, meteorism, no tumor in right lower quadrant, no mass felt per rectum, melena, duration, five days; operation: no abdominal exudate, diverticulum contained no tumor, was gangrenous and was reducible, spinal anæsthesia; died on operating table.

CASE XXX.—Boulay,[9] 1920, age eleven years, female; present illness: pain, vomiting, no meteorism, tenderness, no tumor in right lower quadrant, no mass felt per rectum, temperature 37.5° C., melena, duration, thirty hours; operation: abdominal exudate, diverticulum contained no tumor, was not gangrenous and was not reducible, resection of bowel; recovery.

CASE XXXI.—Fabre,[39] 1920, age twelve years, female, previous abdominal distress; present illness: colicky pain, vomiting, no meteorism, tenderness, tumor in right lower

quadrant, temperature 38.5° C., duration, thirty hours, operation: diverticulum contained no tumor, was not gangrenous and was not reducible, resection of bowel; death.

CASE XXXII.—Coutts,[23] 1920, age four years, male; present illness: pain, tenderness, rigidity, tumor in right lower quadrant, melena; operation: diverticulum contained a tumor, was not gangrenous and was reducible, resection of diverticulum only; recovery.

CASE XXXIII.—King,[76] 1921, age twenty-six years, male, previous attack two months before; present illness: pain, operation: ileum perforated with much abdominal exudate, diverticulum not reducible, resection of bowel; recovery.

CASE XXXIV.—Fröhlich,[43] 1921, age four years, male, daily pain for eight days, two days before operation fell on abdomen, six hours later attack began; present illness: pain, vomiting, no meteorism, rigidity, tumor in right lower quadrant, no melena, duration, two days; operation: diverticulum contained a tumor and was not reducible, resection of bowel; death.

CASE XXXV.—Depisch,[27] 1921, age twenty-five years, female, abdominal pain two years before; present illness: colicky pain, vomiting, no meteorism, no tenderness, no rigidity, no tumor in right lower quadrant, no mass felt per rectum, temperature 37.6° C., no melena, duration, thirty hours; operation: abdominal exudation, diverticulum contained a tumor, was gangrenous and was reducible, resection of bowel; recovery.

CASE XXXVI.—Wienecke,[136] 1921, age two years, male, attack a few weeks before; present illness: pain, vomiting, visible peristalsis, no meteorism, no tenderness, no rigidity, temperature 37.5° C., melena, duration, thirty-six hours; operation: abdominal exudate, diverticulum contained no tumor, was not gangrenous and was reducible, resection of bowel; recovery.

CASE XXXVII.—Hood,[60] 1923, age eight years, male, abdominal pain several years before; present illness: pain, vomiting, tumor in right lower quadrant, no mass felt per rectum, temperature 37.2° C., melena, duration, eighteen hours; operation: diverticulum contained no tumor, was gangrenous and not reducible, resection of bowel; recovery.

CASE XXXVIII.—Vickers,[130] 1923, age seven years, male; present illness: colicky pain, vomiting, meteorism, tumor in left lower quadrant, melena, duration, thirty-six hours; operation: diverticulum gangrenous but reducible, resection of bowel; recovery.

CASE XXXIX.—Fuchsig,[45] 1923, age eighteen months, male, frequent previous attacks of abdominal pain; present illness: pain, vomiting, no meteorism, tumor above umbilicus, no melena; duration, twelve hours; diverticulum contained a tumor, was gangrenous and was not reducible, resection of bowel; death.

CASE XL.—Fuchsig,[45] 1923, age seventeen years, male, no previous attacks; present illness: pain, meteorism, tenderness, tumor in right lower quadrant, temperature 39.0° C., no melena, duration, five days; operation: diverticulum contained no tumor, was gangrenous and was reducible, resection of bowel; death.

CASE XLI.—Fuchsig,[45] 1923, age nineteen years, male, frequent abdominal pains as a child; present illness: pain, vomiting, meteorism, rigidity, tumor in right lower quadrant, no fever, no melena, duration, six hours; operation: diverticulum contained no tumor, was not gangrenous and was not reducible, resection of bowel; recovery. Second bowel resection several months later because of adhesions.

CASE XLII.—Johnson,[71] 1923, age eight months, male, no previous attacks; present illness: colicky pain, vomiting, meteorism, tenderness, tumor in right lower quadrant, temperature 37.7° C., no melena, duration, thirty-six hours; operation: diverticulum contained no tumor, was gangrenous and was not reducible, resection of bowel; death.

CASE XLIII.—Greenwood,[50] 1923, age thirty years, female, no previous attacks; present illness: colicky pain, vomiting, no meteorism, tenderness, rigidity, tumor in right lower quadrant, mass felt per vaginum, temperature 36.6° C., no melena, duration two days; operation: diverticulum contained no tumor, was gangrenous and was reducible, resection of bowel; recovery.

CASE XLIV.—Stone,[120] 1923, age eighteen months, male, no previous attacks; present illness: pain, vomiting, no meteorism, no tenderness, tumor on left side, no mass felt

per rectum, melena, duration twelve hours; operation: the bowel was not involved in the invagination, only the diverticulum being inverted, diverticulum contained no tumor, was not gangrenous and was reducible, resection of diverticulum only; recovery.

CASE XLV.—Braun and Worthman,[11] 1924, age eighteen months, male; present illness: vomiting, tumor on left side, mass prolapsed from rectum, duration, eight hours; operation: diverticulum reducible, resection of diverticulum only; death.

CASE XLVI.—Lower,[86] 1925, age forty-three years, male, attacks of abdominal pain for one year, three attacks in past week; present illness: colicky pain, vomiting, meteorism, rigidity, tumor in right upper quadrant, no mass felt per rectum, fever, no melena, duration, two hours; operation: diverticulum contained no tumor, was gangrenous and was not reducible, resection of bowel; recovery.

CASE XLVII.—Lower,[86] 1925, age forty-four years, female, attacks of colic for six months; present illness: colicky pain, vomiting, meteorism, tenderness, no rigidity, no tumor in right lower quadrant, no mass felt per rectum, no melena, duration, seven days; operation: diverticulum contained no tumor and was not reducible, resection of bowel; outcome not stated.

CASE XLVIII.—Kaspar,[74] 1925, age fifteen years, male, previous attack three days before with complete recovery; present illness: colicky pain, vomiting, meteorism, tenderness, rigidity, no tumor in right lower quadrant, no melena, duration, three days; operation: abdominal exudate; diverticulum contained a tumor, was gangrenous and was reducible, resection of bowel; recovery.

CASE XLIX.—Mathews,[80] 1925, age thirty years, male; present illness: colicky pain, vomiting, duration, two days; operation: diverticulum was not gangrenous and was reducible, resection of bowel; death.

CASE L.—Wessel,[135] 1926, age thirty-four years, male, abdominal pain for eighteen months; present illness: pain, melena; operation: resection of bowel; recovery.

CASE LI.—Schlutz,[118] 1927, age three months; present illness: meteorism, fever, melena; operation: diverticulum contained no tumor, was not gangrenous and was reducible, resection of bowel; death.

CASE LII.—Mathieu and Davioud,[91] 1927, age seven years, male, painful melena three years before and seven months before; present illness: colicky pain, vomiting, rigidity, no tumor in right lower quadrant, no mass felt per rectum, temperature 37.5° C., melena, duration, twelve hours; operation: abdominal exudate, diverticulum contained a tumor, was not gangrenous and was not reducible, resection of bowel; recovery.

CASE LIII.—Mathieu and Davioud,[91] 1927, age one year, male; present illness: melena of sixty hours' duration; operation: diverticulum reducible, simple disinvagination, no resection; death.

CASE LIV.—Picot,[110] 1927, age thirteen years, male, colic since birth; present illness: colicky pain, vomiting, no meteorism, tenderness, no rigidity, tumor in right lower quadrant, no melena, duration, thirty-six hours; operation: diverticulum contained a tumor, was not gangrenous and was reducible, resection of diverticulum only; recovery.

CASE LV.—Holst,[65] 1928, age twelve years, male, attacks of abdominal pain for five months; present illness: pain, tenderness, rigidity, no tumor in right lower quadrant, temperature 38.0° C., duration, twenty-six hours; operation: abdominal exudate, diverticulum contained a tumor, was not gangrenous and was reducible, resection of diverticulum only; recovery.

CASE LVI.—Montgomery,[97] 1928, age two years, female, no previous abdominal distress; present illness: pain, vomiting, no meteorism, tenderness, tumor in right lower quadrant, no mass felt per rectum, melena, duration, one day; operation: diverticulum contained a tumor, was not gangrenous and was reducible, simple disinvagination, no resection; death.

CASE LVII.—Eisberg,[24] 1928, age eleven years, male, abdominal pain one year before; present illness: colicky pain, vomiting, no meteorism, tenderness, tumor in right lower quadrant, no mass felt per rectum, no melena, duration, thirty-six hours; operation:

abdominal exudate, diverticulum contained a tumor, was gangrenous and was not reducible, intussusception was double, resection of bowel; recovery.

CASE LVIII.—Pedersen,[107] 1928, age two years, male, umbilical hernia since birth; present illness: melena of one day's duration; operation: diverticulum gangrenous, no resection, anastomosis only; death.

CASE LIX.—Pedersen,[107] 1928, age eight years, male; present illness: colicky pain of two days' duration; operation: abdominal exudate, diverticulum reducible and not gangrenous, resection of diverticulum only; recovery.

CASE LX.—Pedersen,[107] 1928, age twenty years, male; present illness: pain and melena of three days' duration; operation: abdominal exudate, diverticulum gangrenous and not reducible, resection of bowel; recovery.

CASE LXI.—Decker,[25] 1928, age seven years, male, no previous abdominal crises; present illness: colicky pain, vomiting, meteorism, tenderness, rigidity, tumor in right lower quadrant, no mass felt per rectum, temperature 37.5° C., melena, duration, three days; operation: abdominal exudate, diverticulum reducible and not gangrenous, resection of diverticulum only; death.

CASE LXII.—Macdonald,[97] 1928, age forty years, male, no previous abdominal distress; present illness: colicky pain, vomiting, meteorism, no rigidity, no tumor in right lower quadrant, no mass felt per rectum, no melena, duration, fourteen days; operation: diverticulum contained a tumor, was not gangrenous and was not reducible, resection of bowel and colostomy; recovery.

CASE LXIII.—Pettersen,[109] 1928, age seven years, female; present illness: colicky pain, tenderness, no tumor in right lower quadrant, no mass felt per rectum, temperature 38.3° C., duration, two days; operation: abdominal exudate, diverticulum contained a tumor, was not gangrenous and was reducible, resection of diverticulum only; recovery.

CASE LXIV.—Cannon,[16] 1928, age eighteen months, female; operation: diverticulum not gangrenous and reducible, resection of diverticulum only; recovery.

CASE LXV.—Allman,[3] 1928, age eight years, male, no previous abdominal distress; present illness: colicky pain, vomiting, meteorism, rigidity, no tumor in right lower quadrant, temperature 37.6° C., no melena, duration, three days; operation: abdominal exudate, diverticulum contained no tumor, was gangrenous and was reducible, resection of diverticulum only; recovery.

CASE LXVI.—McIver,[94] 1928, age twelve years, male, abdominal pain eight months before; present illness: colicky pain, vomiting, no meteorism, tenderness, rigidity, no tumor in right lower quadrant, no mass felt per rectum, temperature 40.0° C., no melena, duration, forty hours; operation: abdominal exudate, diverticulum contained no tumor, was gangrenous and not reducible, bowel resection and colostomy; death.

CASE LXVII.—Doolin,[30] 1929, age forty-four years, male, operation five days before for right inguinal hernia at which time a normal Meckel's diverticulum was seen in the hernial sac; onset of present illness with sudden onset of colicky pain, vomiting, tumor in right lower quadrant, and melena, duration, three hours; operation: diverticulum contained no tumor, was not gangrenous and was not reducible, resection of diverticulum only from within the bowel; recovery.

CASE LXVIII.—Livingston,[86] 1929, age eighteen months, male; present illness: colicky pain, vomiting, no rigidity, tumor in right lower quadrant, no mass felt per rectum, no melena, duration, six hours; operation: diverticulum contained no tumor, was not gangrenous and all but its tip was reducible, resection of diverticulum only; recovery.

CASE LXIX.—Christopher,[20] 1930, age nine months, male, no previous attacks; present illness: colicky pain, vomiting, tumor in right lower quadrant, no mass felt per rectum, melena, duration, ten hours; operation: abdominal exudate, diverticulum contained no tumor, was gangrenous and was reducible, disinvagination followed by ileostomy several days later; death.

CASE LXX.—Lenner,[83] 1930, age six months, male; present illness: pain, vomiting, meteorism, tenderness, no rigidity, no tumor in right lower quadrant, no mass felt per rectum, much melena, duration, three days; operation: diverticulum contained no tumor, was gangrenous and was reducible; resection of diverticulum only with ileocolic anastomosis; death.

CASE LXXI.—Trask and Turvey,[135] 1921, age one year, male, no previous attacks; present illness: no pain, no colic, vomiting, meteorism, no tenderness, no rigidity, tumor in right lower quadrant, no mass felt per rectum, no fever, no melena, duration, four days; operation: diverticulum contained no tumor and was gangrenous, ileostomy; result not stated.

CASE LXXII.—Andrews,[4] 1932. Vide supra.

CASE LXXIII.—Harkins, 1932. Vide supra.

CASES LXXIV to CXIV.—Hertzler and Gibson,[61] 1913, average age thirteen years in forty cases with data given; thirty-one males, seven females in thirty-eight cases with data given; previous attacks, seventeen out of twenty cases with data given; pain, thirty-one out of thirty-one cases with data given; vomiting twenty-seven out of twenty-seven cases with data given; meteorism, seventeen out of twenty-two cases with data given; tenderness, ten out of fifteen cases with data given; rigidity, four out of nine cases with data given; tumors in right lower quadrant, sixteen out of twenty-four cases with data given; mass felt per rectum, two out of five cases with data given; colic, sixteen out of sixteen cases with data given; fever three out of seven cases with data given; melena, twelve out of eighteen cases with data given; duration of attack, average 132 hours in twenty-two cases with data given; abdominal exudate, ten out of eleven cases with data given; tumor of diverticulum, five out of six cases with data given; gangrene, eleven out of twelve cases with data given; diverticulum reducible, eleven out of twenty-five cases with data given; resection of diverticulum only, eleven out of forty-one cases with data given; resection of bowel, twenty-three out of forty-one cases with data given; and recovery, twenty-one out of forty-one cases with data given.

REPORTED CASES: GROUP II

Series of forty-six cases with such incomplete data that they cannot be included in the analysis of data. The division between Groups I and II is of necessity somewhat arbitrary. Reports of intussusception of a Meckel's diverticulum that has prolapsed from the umbilicus such as the case of Peters, or the four cases of Corner,[22] are not included in either Group I or Group II.

CASE CXV.—Pathological specimen in St. Bartholomew's Hospital Anatomical Museum, 1846. Male, aged thirty-six. The patient had attacks of abdominal pain for six months before death from intestinal obstruction and peritonitis. The specimen is that of a somewhat gangrenous ileocolic intussusception with a diverticulum of the ileum at the apex.

CASE CXVI.—Treves,[127] 1884, pathological specimen.

CASE CXVII.—Heller,[60] 1885, male, aged seventy, pathological specimen.

CASE CXVIII.—Heller,[60] 1885, male, aged sixty-six; death from pyonephrosis; pathological specimen.

CASE CXIX.—Ingle,[68] 1888, child, aged five months, with vomiting, abdominal pain, tympanites, hiccough, and bloody mucus in the rectum. There was no tumor or tenderness evident on palpation. No operation. Death on the fifth day. Pathological specimen.

CASE CXX.—Adams,[2] 1891, male, with obscure symptoms of intestinal strangulation for two weeks. No operation. Pathological specimen.

CASE CXXI.—Willett,[137] 1891, male. Pathological specimen.

CASE CXXII.—Heller,[60] 1891, male, aged forty-six. Death from perforated peptic ulcer. Necropsy showed an invaginated Meckel's diverticulum with an accessory pancreas at its tip.

CASE CXXIII.—O'Connor,[105] 1894, male, aged thirteen, with absolute constipation for eight days and pain for two days, passed per rectum an invaginated portion of intestine eleven inches long with an attached Meckel's diverticulum. The patient was alive and well a year later.

CASE CXXIV.—McFarland,[93] 1901, male, aged sixty; death from intestinal obstruction and uræmia. Necropsy showed purulent peritonitis and a Meckel's diverticulum four centimetres long rotated 90° and invaginated for about two centimetres into the ileum, producing gangrene and partial obstruction.

CASE CXXV.—Smith,[119] 1903, pathological specimen.

CASE CXXVI.—Treves,[127] 1903, pathological specimen.

CASE CXXVII.—Porter,[112] 1905, male, aged three, with symptoms of complete intestinal obstruction. Death during laparotomy. The child was moribund before operation. An invaginated ileum and invaginated Meckel's diverticulum were found.

CASE CXXVIII.—Hirschsprung,[63] 1905, male, aged seven, with symptoms of ileus followed by the spontaneous passing of a portion of small bowel with an inverted diverticulum at the end. Death five weeks later from a perforation at the site of the cicatrix.

CASE CXXIX.—Hirschsprung,[63] 1905, male, aged five months. A resection of the bowel was performed at the site of an ileal intussusception with inverted Meckel's diverticulum of seventeen hours' duration; death.

CASE CXXX.—Jepson,[70] 1905, child, aged sixteen months. At operation the intussuscepted ileum and attached Meckel's diverticulum were readily disinvaginated. Owing to the child's grave condition, the diverticulum was not removed. Three years later there had been no recurrence.

CASES CXXXI to CXXXVI.—Hess,[62] 1905. In a series of 314 instances of intussusception collected from the literature, six were due to an invaginated Meckel's diverticulum.

CASE CXXXVII.—Guyot,[55] 1907, child, aged ten. Operation was performed the fifth day of an evident intestinal obstruction. Disinvagination was impossible and ileostomy was performed. Death occurred the next day. Necropsy revealed a gangrenous ileal intussusception with an invaginated Meckel's diverticulum.

CASE CXXXVIII.—Kingsford,[77] 1909, male, aged ten. Intussusception was diagnosed, but operation not permitted. At necropsy, an intussusception was found with a Meckel's diverticulum at its tip that was not inverted.

CASE CXXXIX.—Müller,[103] 1909, male, aged thirty-six. After an acute onset of pain in the cæcal region, the patient had constipation, rectal tenesmus, generalized abdominal tenderness, and moderate distention for three weeks. He then passed per rectum a slough of intestine with an invaginated Meckel's diverticulum. Death occurred two weeks later from perforative peritonitis.

CASES CXL to CXLIII.—Walton,[133] 1911. In the years 1901 to 1911 inclusive, 864 cases of intestinal obstruction were admitted to the London Hospital. Of these, 239 or 27.6 per cent. were due to intussusception, and in turn, of the 239, four were due to an invaginated Meckel's diverticulum.

CASE CXLIV.—Neumann,[104] 1912, male, aged thirty. At operation an ileal intussusception was found with a Meckel's diverticulum at its apex which was disinvaginated. No resection; recovery.

CASES CXLV to CLI.—Koch and Oerum,[79] 1912. Of 400 cases of intussusception seen in the Copenhagen hospitals, seven were due to an invaginated Meckel's diverticulum.

CASE CLII.—Coutts,[23] 1920, male, aged ten months. The patient had severe colicky pain in the abdomen, vomiting, and a tumor palpable to the left of and above the umbilicus when the patient was anæsthetized. Operation performed the day after the onset revealed an intussusception that was disinvaginated. At the apex of the intussusception was a cyst two centimetres in diameter which was removed. Death occurred from broncho-

pneumonia. The cyst was a few inches oralwards from the ileocæcal valve and was believed to be a remnant of a Meckel's diverticulum.

CASES CLIII to CLVII.—Perrin and Lindsay,[108] 1921. Of 400 cases of intussusception seen in the London Hospital from 1903 to 1920, five were due to an invaginated Meckel's diverticulum.

CASE CLVIII.—Braun and Worthman,[11] 1924, male, aged thirty, with a fifteen-centimetre long ileal intussusception and invaginated Meckel's diverticulum. Resection of the diverticulum only; recovery.

CASES CLIX and CLX.—McIver,[94] 1928. In a series of thirteen ileal intussusceptions at the Massachusetts General Hospital, three were caused by an invaginated Meckel's diverticulum. (See also Case LXVI.)

ANALYSIS OF THE LITERATURE.—On the basis of the 114 cases in Group I, an analysis is made of the symptoms, course and treatment of intussusception due to invaginated Meckel's diverticulum. In analyzing a large number of cases reported by various authors, it must be remembered that a statistical survey has many limitations. Authors are apt to record only positive findings; thus, of forty-three cases where data are given, an abdominal exudate is found thirty-nine times. This indicates that an abdominal exudate is present in either 90 per cent. or 34 per cent. of cases, depending on whether forty-three or the total 114 cases are made the basis of calculation. For this reason the use of percentage has been avoided as much as possible. Throughout the following analysis, it is to be remembered that 114 cases of intussusception due to invaginated Meckel's diverticulum are under consideration.

OCCURRENCE.—*Occurrence of Meckel's diverticulum in normal persons.*—In 10,360 necropsies, Turner[128] found eighty-one instances of Meckel's diverticulum (0.8 per cent.). Forgue and Riche[42] collect statistics of 112 instances of Meckel's diverticulum in 7,850 necropsies (1.4 per cent.). Cunningham's[24] Anatomy states that it was present in seventy-three of 3,302 bodies (2.2 per cent.). Kelynack[75] found it in eighteen of 1,446 bodies (1.2 per cent.). Mitchell[96] found thirty-nine in 1,635 necropsies (2.4 per cent.). Knox[78] found three in 500 necropsies (0.6 per cent.). Schaetz[117] found seventeen in 737 bodies. In 2,400 necropsies at the University of Chicago* from 1902 to 1931, forty-one instances of Meckel's diverticulum were found (1.7 per cent.); thirty-one of these were in males and ten in females. The ages varied between one day and eighty-six years. In no instance was the diverticulum the cause of death.

Combining my own series with those of Turner,[128] Forgue and Riche[42] (who include the data of Kelynack[75] and Mitchell[96]), Cunningham,[24] Knox,[78] and Schaetz,[117] there are 327 instances of Meckel's diverticulum in 25,149 necropsies, or 1.3 per cent. This agrees with the operative statistics of Harbin,[69] who found seven instances of Meckel's diverticulum in 507 consecutive laparotomies where a routine exploration was performed (1.4 per cent.). In Balfour's[5] statistics of fifteen cases of Meckel's diverticulum in 11,107 laparotomies (0.1 per cent.), the incidence was lower because a routine exploration was not performed.

The proportion of all cases of intussusception that is caused by a Meckel's diverticulum.—Fitzwilliams[41] finds fifteen cases of the Meckel's diverticulum type of intussusception in a series of 1,000 intussusceptions. Eliot and Corscaden,[35] twenty-nine out of 300; Koch and Oerum,[79] seven out of 400;

*I am indebted to Dr. Esmond Long for permission to publish these figures.

TABLE I

Incidence of Meckel's Diverticulum from Necropsy Figures

Author	No. of Necropsies	No. of Cases of Meckel's Diverticulum	Percentage Incidence of Meckel's Diverticulum
Turner	10,360	81	0.8
Forgue and Riche	7,850	112	1.4
Cunningham	3,302	73	2.2
Knox	500	3	0.6
Schaetz	737	17	2.3
Harkins	2,400	41	1.7
Total	25,149	327	1.3

Perrin and Lindsay,[108] five out of 400; and Hess,[62] six out of 314. From these collections, it seems that a Meckel's diverticulum is a factor in about 2.5 per cent. of intussusceptions. This figure is probably too high because Fitzwilliams[41] and Eliot and Corscaden[35] collected their cases from the literature where rare cases are more likely to be reported and 1.5 per cent. probably represents a more accurate figure. This is practically the same as the incidence of Meckel's diverticulum in normal persons. From these statistics it appears that persons with a Meckel's diverticulum may not be more likely to develop intussusception than normal individuals, but if an intussusception does develop, it does so at the site of the Meckel's diverticulum.

Relationship of intussusceptions due to Meckel's diverticulum and other diseases of Meckel's diverticulum.—Porter[112] found twenty cases of intussusception in a series of 184 affections of Meckel's diverticulum. Forgue and Riche[42] found thirty-four intussusceptions in 287 cases of obstruction due to the organ; Drummond[32] found seven out of twenty-two; and Wellington,[134] fifty nine out of 326. Thus it would seem that 17 per cent. of the Meckel's diverticula that cause trouble do so by producing intussusception. Halstead[58] finds that 6 per cent. of 991 cases of acute intestinal obstruction were due to a Meckel's diverticulum.

GEOGRAPHICAL DISTRIBUTION

Various authors, including Fuchsig[45] and Koch and Oerum,[79] have pointed out that intussusception is most commonly reported in England, Denmark, Australia, and the United States, and is rarely seen in Germany and Austria. No figures are available as to the geographical distribution of Meckel's diverticulum. In the series of the Meckel's diverticulum type of intussusception collected in the present paper, the distribution seems to follow somewhat that of intussusception. Of the 160 cases, England reported forty-seven; Germany, forty-one; the United States, twenty-eight; Denmark, eighteen; France, sixteen; Sweden, three; and Russia, Spain, Switzerland, and Australia, each one.

AGE.—*Age of patients with intussusception of all types.*—Hess[62] found

that 64 per cent. of his 314 cases of intussusception occurred under the age of one year. Other authors reported the following percentages as occurring under one year: 60 per cent. (Koch and Oerum; of these, two-thirds were between five and seven months of age); 70 per cent. (Perrin and Lindsay; of these, three-fourths were between five and nine months of age); 72 per cent. (Walton[132]); and 72 per cent. (Fitzwilliams). Thus it is seen that most ordinary intussusceptions occur below the age of one year, and of these, a large proportion occur in the period between five and nine months of age.

Age of patients with affections of Meckel's diverticulum of all types.—The average of Porter's series was twenty-one years. Only 28 per cent. of Forgue and Riche's patients were afflicted in the first decade of life.

Age of patients with intussusception due to Meckel's diverticulum.—The average age of the patients in the present series is 13.1 years. The forty cases collected by Hertzler and Gibson[61] averaged 13.0 years. Only six of the cases in the present series were below one year of age. In Table II is given the age distribution by decades, the age limits being three months and forty-nine years.

TABLE II

Age Distribution by Decades of Patients with Intussusception Due to Invaginated Meckel's Diverticulum

Age in years.....	0–10	11–20	21–30	31–40	41–50
Number of cases....	60	24	15	7	5

It is seen that the age distribution of the Meckel's diverticulum type of intussusception does not agree at all with that of intussusception as a whole. It agrees somewhat with that of other affections of Meckel's diverticulum. In reviewing 300 cases of intussusception in adults, Eliot and Corscaden[35] found the enteric variety more common than in children. McIver found all of his thirteen cases of enteric intussusception were over thirteen years of age. Perrin and Lindsay have graphed separately the age distribution of the various types of intussusception, finding that the ileocæcal and ileocolic types have the usual age-distribution curve, being much more common in infants, while the colic does to a slight extent, and the enteric has little relation to age. Thus it seems that the age distribution of the Meckel's diverticulum type of intussusception is similar to enteric intussusception.

SEX.—*Sex of patients with intussusception of all types.*—Sixty-eight per cent. of Fitzwilliam's cases of intussusception, 69 per cent. of Koch and Oerum's cases, and 64 per cent. of Perrin and Lindsay's cases are males.

Sex of patients with affections of Meckel's diverticulum of all types.—The percentage of males as given by various authors is as follows: 75 per cent. (Porter); 76 per cent. (Wellington); and 82 per cent. (Forgue and Riche). In the University of Chicago necropsy series, in no cases of which was there any affection of the Meckel's diverticulum, 76 per cent. were males.

Sex of patients with intussusception due to Meckel's diverticulum.—Since the preponderance of intussusceptions and of Meckel's diverticula when

occurring separately is in males, it is not surprising that the combination attacks males more frequently than females. In 105 cases in the present series where the sex is given, 79 per cent. are males.

Previous abdominal crises.—No data are available as to the proportion of all types of intussusception that have had previous attacks, but in general it is recognized that intussusception is an acute disease with rare recurrences. All writers on intussusception due to Meckel's diverticulum have noted the frequent history of abdominal crises. In the present series, in forty-six cases there was a definite history of previous abdominal crises, in twenty-one cases it was stated that there was no history of previous abdominal distress, and in forty-seven cases no data were given.

Duration of the final attack.—Closely related to the history of previous abdominal distress is the duration of symptoms of the final attack. In many of the cases reviewed for this paper, the symptoms were of such long duration and of such remittency that it was difficult to tell exactly when the present illness began. Furthermore, since most of the cases were terminated, successfully or otherwise, by operative intervention, the duration of the final attack was as much dependent upon the impression of necessity for operation that the disease aroused in the minds of the surgeons as it was upon the severity of the disease itself. A surgeon often tends to delay where he cannot make a definite diagnosis, even though operative indications are present. Thus there is a psychological element that may affect the duration of the attack that is terminated by operation. In both the patients operated upon at the University of Chicago, and in a considerable number of the cases reviewed, there was a less severe attack a day or two before the final severe attack. Some authors believe this is due to the inversion of the diverticulum before the bowel becomes invaginated. Perrin and Lindsay found the average duration of 156 ileocæcal intussusceptions was thirty-eight hours, of 126 ileocolic intussusceptions was thirty-three hours, and of twenty-seven enteric intussusceptions was eighty-four hours. The cases due to Meckel's diverticulum agree much more closely with this last figure. Corner points out that "as the origin of the invagination is lateral, the ring where the bowel is infolded is very oblique, and consequently the intestinal lumen is not completely occluded, and the case may be clinically subacute. This is a very interesting pathological fact, and explains why some of the recorded cases give long subacute histories." In the 114 cases, of eighty-eight cases where the duration of the final attack was given, the average was 81.6 hours. Some of the earlier cases were allowed to go longer before operation because Hertzler and Gibson's portion of the series, all reported before 1912, averaged 132 hours. The remainder averaged 64.3 hours, reducing the grand average as stated before to 81.6 hours.

Pain.—Pain was present in 76 per cent. of the cases under one year and 85 per cent. of those over one year in Koch and Oerum's series of intussusception of all types. In the present series of intussusception due to Meckel's diverticulum, pain was present in ninety-seven cases, definitely stated to be

absent in one case, and no data were given in sixteen cases. The pain was quite often of a colicky type. In the present series, it was stated to be colicky fifty-two times, stated not to be colicky twice, and in sixty instances no data were given on this point. One of the University of Chicago patients (Case LXXIII) had a very definite colic, occurring every seven to eight minutes with almost complete relief between attacks. The fact that the patients are older in the type of intussusception due to Meckel's diverticulum than in ordinary intussusception may explain the more frequent recording of pain. When intussusception of the large bowel occurs, the mesentery is not necessarily dragged along with the intussusception, but in enteric intussusceptions, whether or not due to a Meckel's diverticulum, the mesentery is usually dragged into the infolding and the traction may be one of the chief elements in the more frequent pain in this type of intussusception.

Vomiting.—In Koch and Oerum's series of intussusception of all types, vomiting was present in 92 per cent. of the cases under one year and 91 per cent. over one year. In Hess' series of 314 cases, vomiting was present in 166 and absent in four. Since the obstruction in the Meckel's diverticulum variety of intussusception is higher in the intestinal tract, one might expect vomiting to be more frequent than in ordinary intussusception. In the present series, vomiting was present eighty-two times, in no case was it definitely stated to be absent, and data were not given on this point thirty-two times. In the two University of Chicago patients, vomiting was out of all proportion to the other symptoms.

Meteorism.—Since the obstruction due to the Meckel's diverticulum type is higher than in ordinary intussusception, more vomiting and less meteorism would be expected. This might be equalized to some extent in that the Meckel's diverticulum variety of intussusception is less readily diagnosed and allowed to run a longer course before operation. In the present series, meteorism was stated to be present forty-five times, absent twenty-four times, and forty-five times no statement concerning it was made.

Tenderness and rigidity.—These signs are present in about an equal proportion of all intussusceptions. In the present series, tenderness was present forty-one times, absent thirteen times, and no data were given sixty times. Rigidity was present twenty times, absent twenty-two times, and no data were given seventy-two times. In several cases there was no tenderness or rigidity in spite of well-advanced gangrene. Tenderness, rigidity, and abdominal exudate may be indicative of the extent of injury to the intestinal outer coats, including the peritoneum, while blood in the stools gives similar evidence of the state of nutrition of the mucous membranes.

Abdominal tumor.—Statistics show that in ordinary intussusception a tumor is palpable abdominally in the following proportion of cases: 63 per cent. (Perrin and Lindsay); 83 per cent. (Eccles[33]); 93 per cent. (Hess); and 85 per cent. (Koch and Oerum). The last-named authors find a tumor equally frequent in patients below and above one year of age. They also state

that the mass is usually found in the left upper abdominal quadrant due to wandering of the intussusception. Hess states that in ninety-four patients where the position of the tumor was noted, it was on the left side in forty-three instances as opposed to twenty-three on the right. The type of intussusception due to Meckel's diverticulum starts near the cæcum and the tip of the intussusception seldom proceeds beyond the hepatic flexure. Gogibus[48] states that in only 11 per cent. of this type of intussusception does the apex reach as far as the transverse colon. Thus, a mass if felt at all would be expected to be on the right side. In the present series, a mass was palpable in the right lower abdominal quadrant forty-one times, in the mid-line or the right upper abdominal quadrant three times, and only five times on the left side. Thirty-three case histories contained no statement on this point.

Tumor palpable per rectum.—Since the Meckel's diverticulum type of intussusception seldom advances beyond the hepatic flexure, it would be unlikely that a tumor would be palpable per rectum. In only thirty-seven of the 114 cases was it stated that a rectal examination was made. In only four of these was a tumor felt per rectum and two of these were definitely stated to be outside the bowel. On the contrary, in ordinary intussusception, a mass is palpable per rectum rather frequently: 48 per cent. (Hess); 40 per cent. under one year of age and 27 per cent. over one year of age (Koch and Oerum); and 27 per cent. in ileocæcal intussusceptions and 13 per cent. in ileocolic intussusceptions (Perrin and Lindsay).

Melena.—It is probable that all intussusceptions produce some melena, but in general the type of intussusception caused by Meckel's diverticulum does not produce as much as the ordinary variety. Statistics as to the occurrence of melena in ordinary intussusception are as follows: 89 per cent. (Perrin and Lindsay); 89 per cent. (Eccles); 97 per cent. of cases in which symptom was recorded (Hess); 95 per cent. under one year of age and 75 per cent. over one year (Koch and Oerum); and 94 per cent. in children under two years of age, 62 per cent. in larger children, and only 38 per cent. in adults (Braun and Worthman). In the present series, forty-three patients had melena, thirty-four did not, and in thirty-seven no data were given. Fuchsig says that the diverticulum may swell and plug the intestine and prevent marked melena.

Another source of melena that must be borne in mind is from ulcers adjoining gastric mucosa in a Meckel's diverticulum. Schaetz found gastric mucosa in five out of thirty Meckel's diverticula found at necropsy (16.6 per cent.). All of these patients were males and died of some other disorder, the Meckel's diverticulum being an incidental finding. Lindau and Wulff[84] report thirty-seven instances of gastric mucosa in Meckel's diverticulum with microscopical verification. Seventy-seven per cent. of these patients were males. These represent findings at operation, ulceration being present in all cases, perforation in about half and a history of severe hæmorrhage or anæmia being present in most of them. Unlike intussusception with invaginated Meckel's diverticulum, the hæmorrhage in these cases is not accom-

panied by signs of intussusception, the bleeding is apt to be more profuse, the blood may produce a tarry stool, mucus is usually not present, and occasionally there may be a burning pain similar to that produced by ulcer elsewhere. Stone's patient (Case XLIV in my series) had gastric mucosa in an invaginated Meckel's diverticulum. There was no ulceration of the mucosa and the symptoms did not differ radically from other cases in my series.

Fever.—Hess states that fever occurs in 40 per cent. of all cases of invagination in which the symptom is referred to, usually early in the attack. However, Braun and Worthman state that of thirty-three patients with ordinary intussusception, only three had fever above 38.0° C. and only seven, any fever at all. In the present series of the Meckel's diverticulum variety of intussusception, fever seems to be insignificant. Thirty-nine patients had their temperature recorded in the case histories. Of these only fifteen had a fever over 37.5° C., and only six of these were over 38.0° C. One patient was moribund with a temperature of 35.0° C. The white blood count was given on only eight cases, being 11,000 (Andrews); 16,000 (Harkins); 14,000 (Johnson); 7,400 (Trask and Turvey); 16,000 (Decker); 27,000 (McIver); 22,000 (Fauntleroy); and 5,000 (Allman).

Abdominal exudate.—An interesting finding in thirty-nine of forty-three cases where data are given, is that of considerable fluid in the peritoneal cavity. In several cases this has been large enough in amount so that it might have been determined by percussion before operation.

Tumor of the diverticulum.—An interesting feature of twenty-six of the 114 cases is that a tumor was present at the tip of the Meckel's diverticulum. In 1925, Kaspar found thirty-one instances of tumor of Meckel's diverticulum. Fourteen, or almost half, of these had produced intussusception. This group formed about 20 per cent. of Kaspar's series of seventy-two cases of intussusception due to Meckel's diverticulum. Since 1925, I find records of seven additional instances of tumor of Meckel's diverticulum, including a papilloma (Picot) and an accessory pancreas (Mathieu and Davioud). Recently Michael and Bell[95] collected eleven cases of malignant tumor of Meckel's diverticulum and reported the first instance of adeno-carcinoma of the organ. Thus, about forty-nine tumors of Meckel's diverticulum are on record, almost a third of which contain pancreatic tissue, and over half of which have produced intussusception. Bize postulates that in these cases the diverticulum may not be of the Meckel's type, but due to traction by the tumor, similar to the traction diverticula in the œsophagus. In the University of Chicago necropsy series of forty-one Meckel's diverticula, there is no recorded instance of tumor of the organ.

Miscellaneous Features.—*Invagination of the diverticulum only.*—Several of the patients, including those of Cheyne, Mothersole, McFarland, Ewald, Maroni, Küttner and two cases of Heller, had invagination of the diverticulum only. Cheyne's patient had only the mucous membrane of the diverticulum invaginated. Hohlbeck's[64] patient had a complete intussusception

of the bowel, but of the diverticulum, only the mucous membrane was invaginated. In general the symptoms and signs of these cases are similar to those where the entire bowel is intussuscepted. All cases may begin with a primary invagination of the diverticulum. In one patient, the diverticulum was not invaginated even though it formed the apex of the intussusception.

Umbilical cicatrix.—Three of Drummond's seven patients had abnormal or cicatricized umbilicuses. Gray's patient had an umbilical cicatrix, while Bidwell's[7] patient and Pedersen's first patient had umbilical herniæ. Other authors do not point out such a high frequency of umbilical lesions.

Spontaneous passage of separated intussusceptum per rectum.—The patients of O'Connor, Hirschsprung (second case), and Müller passed an intussusceptum with an attached Meckel's diverticulum. Two of these patients died later from perforation at the site of the cicatrix in the bowel. The phenomenon of spontaneous passage of intussusceptum is not rare in ordinary intussusception. One of Walton's 239 ordinary intussusceptions passed the tumor spontaneously. Of Eliot and Corscaden's collection of 300 cases of intussusception in adults, forty-three passed a necrotic intussusceptum per rectum, two of these having attached Meckel's diverticula.

Pathogenesis.—The pathogenesis of intussusception as a whole has been extensively discussed by many authors, especially by Hess. The type of intussusception associated with Meckel's diverticulum and with other tumors seems to have a definite causative factor. Almost always the diverticulum is situated at the apex of the intussusceptum. The chief subject of argument has been whether the diverticulum invaginates primarily or secondarily. Gray believes that the invagination of the diverticulum begins at its base and not at the apex. Except for the one instance where the diverticulum that formed the apex of the intussusceptum was not itself inverted, the bulk of evidence favors the view that the invagination of the diverticulum is primary.

THERAPY.—*Type of therapy.*—Manifestly, no statistics can ever be advanced to test the medical treatment of intussusception due to Meckel's diverticulum, since the condition can be diagnosed definitely only at laparotomy or necropsy. Judging from ordinary intussusception, surgery is indicated. The only question that remains is the type of operation that is advisable. In general, four types of procedure may be followed: (1) Simple disinvagination; (2) additional resection of the diverticulum; (3) bowel resection; and (4) miscellaneous procedures such as colostomy, Mikulicz colostomy, obliteration of the lumen of the diverticulum, *etc.* In Table III are presented the results of these various methods. From the table it seems apparent that simple disinvagination, or resection of the diverticulum only are the procedures of choice.

When the bowel is not gangrenous and the tumor is reducible.—In twenty-three instances there was no gangrene and the intussusception was reducible. Four of these were submitted to bowel resection with 25 per cent. recovery. Seventeen were submitted to simple amputation of the diverticulum with 88 per cent. recovery. Two cases were only disinvaginated with one

TABLE III

Results of the Various Types of Surgical Therapy in Intussusception Due to Invaginated Meckel's Diverticulum

Type of Operation	No Gangrene Present			Gangrene Present			Total		
	Cases	Recovered	Per Cent. Recovered	Cases	Recovered	Per Cent. Recovered	Cases	Recovered	Per Cent. Recovered
Simple disinvagination	7	5	71	0	0	0	7	5	71
Resection of diverticulum	31	24	77	3	2*	67	34	26	76
Resection of bowel	29	19	65	30	14	47	59	33	56
Miscellaneous procedures	4	2	50	4	0	0	8	2	25
Total	71	50	70	37	16	43	108	66	61

* Gangrene of diverticulum only.

death and one recovery. Here again the advantage of conservative measures is manifest. Often a portion of bowel is considered gangrenous merely because it is cyanotic and an unnecessary resection is performed. The surgeon may occasionally err in the other direction and not resect a truly gangrenous portion of the bowel; Case LXIX is an example of this. Perrin and Lindsay collect ten instances of gangrene revealed at necropsy where simple disinvagination was done in ordinary intussusception. It is stated that the mucosa may often be gangrenous when the serosa appears viable.

Removal of the diverticulum.—Bidwell's experience is in favor of removing the diverticulum. This author did not resect the diverticulum and had to do so later because of a recurrence. Although this was the only recurrence in our entire series, of all the cases that survived operation, only five did not have the diverticulum removed. Two of the five had the lumen of the diverticulum obliterated by stitches. Another factor in favor of removal of the diverticulum is that the patients so frequently present a history of previous attacks. It has been said that a Meckel's diverticulum is more of a menace to its possessor than is a vermiform appendix. McDonald[92] finds that of 145 diverticula not presenting lesions, only nine were attached, while of 162 causing trouble, 110 were attached. Thus it is the attached diverticula that are such a menace, and not those that hang free, as do those that cause intussusception.

Bowel resection.—In ordinary intussusceptions, surgeons have learned that resection is very dangerous. This fear is not entirely applicable to intussusception due to Meckel's diverticulum. The difference in the average age of the patients is the deciding factor. Perrin and Lindsay did not have a recovery after resection in any patient under three years of age, and could find only eight reports of successful resection of the bowel under one year of age. Dowd[31] finds reports of only six cases with successful bowel resec-

tion in patients younger than one year. In the present series, Greenwood's patient was the only infant under three years of age who successfully withstood a bowel resection. This patient was eight months old.

Prognosis.—In the present series, 59 per cent. of 112 cases recovered. Hess states that in ordinary intussusception, of 314 cases, 211 recovered, and only 103 died. Seventy-four of these cases had a laparotomy and simple disinvagination with only five deaths. There were thirty-eight resections with seventeen recoveries. Koch and Oerum state that the operative treatment of ordinary intussusception gave 74 per cent. mortality below one year of age and 38 per cent. over one year. Thus it seems that the prognosis is worse in intussusception due to invaginated Meckel's diverticulum than it is in ordinary intussusception. In the present series, of thirty-two patients under five years of age, the death rate was 56 per cent., while of seventy-nine patients, five years of age or over, the death rate was 35 per cent. Thus the fact that the patients in the present series are older than those with ordinary intussusception would tend to improve the prognosis, rather than to make it less hopeful.

The one explanation for the high mortality in patients with intussusception due to invaginated Meckel's diverticulum is that the condition is not diagnosed soon enough and operation is done only as a last resort. This is something that can be changed and the main purpose of this paper is that it may enable earlier diagnoses to be made. Hess points out very strikingly that early operation is best in all types of intussusception. Table IV gives the mortality rate of patients operated upon at various periods after the onset. It is evident that the time factor is a very important element in the prognosis, the mortality being almost three times as high when operation is performed after seventy-two hours as when it is performed before twelve hours have elapsed.

TABLE IV

The Benefit of Early Operation in Intussusception Due to Invaginated Meckel's Diverticulum

Number of hours before operation...	Under 12	12–24	24–48	48–72	Over 72
Number of cases.....	9	12	26	15	24
Mortality percentage.....	22	42	31	47	58

DIFFERENTIAL DIAGNOSIS.—Intussusception due to Meckel's diverticulum must be differentiated from all varieties of acute abdominal emergency. It has been most frequently confused with appendicitis because of the age of the patients, the especial involvement of the right lower abdominal quadrant, and the pain and vomiting. Unlike appendicitis, the pain is apt to be colicky, there is more frequently blood in the stool, and other signs of intussusception may be present. The classical rigidity and tenderness of appendicitis are not so apt to be present. The differentiation from other affections of Meckel's diverticulum is apt to be difficult because in both cases there are usually signs of intestinal obstruction. However, other affections of Meckel's diverticulum do not have the characteristics of an intussusception.

From intussusception.—Just as intussusception due to Meckel's diverticulum may be differentiated from appendicitis and from other affections of Meckel's diverticulum by the presence of signs of intussusception, so it may be distinguished from ordinary intussusception by the fact that these signs are atypical. Intussusception due to Meckel's diverticulum occurs in older individuals, pain and vomiting are more common, and definite melena and a palpable mass per rectum are much rarer. A mass is palpable in the right lower abdominal quadrant if at all, while in ordinary intussusception, a mass is more commonly felt on the left side. A history of previous attacks is very common and the disease has a much more chronic course, operation being performed only as a last resort, or under some other diagnosis. The choice of operation is simple disinvagination with resection of the diverticulum, but when necessary, bowel resection is less dangerous than in ordinary intussusception because the patients are older.

From intussusception in adults.—In adults intussusception is most apt to be of the enteric type, which in turn commonly originates from tumors. Kasemeyer collects 284 instances of bowel invagination from tumors, including eighty-five malignant, 192 benign, and seven with the type not stated. Of the benign tumors forty-two were Meckel's diverticula. The age and sex distribution and symptomatology of these cases as a whole are similar to the portion due to Meckel's diverticulum. Most of them affect the small bowel of persons over ten years of age and about 62 per cent. are males. McIver states that of thirteen patients with enteric intussusception from all causes at the Massachusetts General Hospital, nine had had previous attacks. Thus in adults, it is difficult to say definitely that an intussusception is due to a Meckel's diverticulum and not to some other cause. But since the treatment and its indications are the same, the differential diagnosis is of but academic interest. Occasionally the presence of a cicatricized umbilicus may indicate the probability of a Meckel's diverticulum as the causative factor.

SUMMARY AND CONCLUSIONS

(1) Reports of 160 patients with intussusception due to invaginated Meckel's diverticulum are found in the literature. One hundred fourteen of these are subjected to a statistical analysis

(2) A clinical differentiation is made between this special type of intussusception and ordinary intussusception. Several distinguishing features of the variety of intussusception due to Meckel's diverticulum are:

(*a*) It occurs in older individuals.

(*b*) A history of previous attacks is more frequent.

(*c*) The course of the disease is more chronic and there is often a mild attack a day or two before the onset of the major illness.

(*d*) Vomiting is more intense.

(*e*) If a mass is palpable at all, it is much more apt to be on the right side.

(*f*) A mass is palpable per rectum in a much smaller proportion of cases.

(*g*) Bleeding from the rectum is less profuse.

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SYNOVECTOMY OF THE KNEE-JOINT IN CHRONIC ARTHRITIS

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Although synovectomy has been used as an operative procedure with intermittent frequency for the past half century, it is only in the recent few years that its significance in relation to arthritis has been definitely recognized.

Volkman,[1] in 1877, was the first to describe the removal of the synovial membrane of a joint in a tuberculous patient. Mignon,[2] in 1899, reported the removal of the synovial membrane from the knee of a patient with chronic arthritis with complete restoration of function in the joint. Goldthwait,[3] in 1900, performed a partial synovectomy in a knee-joint. Hoffa,[4] in 1904, advocated the removal of the synovial membrane of the knee-joint and called attention to the influence of the fat pad adipose tissue on the pathology of the knee. He stated that the normal adipose tissue is liable to grow and to produce an inflammatory hyperplasia even after a slight injury to the joint. Abbott,[5] in 1908, described an operation for the removal of the fringes of the synovial membrane of the knee. His operation was a hit-or-miss method of picking up the synovial membrane, through two lateral incisions, clamping it, and cutting it off. The object was to "catch as much of the capsule as possible." Murphy,[6] in 1916, did two capsulectomies on cases of hypertrophic villus synovitis. The cartilages were removed together with the synovial membrane and a flap of facia was interposed beneath the patella to avoid adhesions.

The first real correlation of synovial changes with chronic articular disturbances, and the surgical removal of the synovial membrane, was accomplished by Swett[7] and Jones,[8] in 1923. As a result of this work our attitude towards synovectomy has changed so that those who were not in sympathy with this surgical procedure are now openly accepting it as a sane treatment for chronic recurring monarticular polyarticular arthritis. Among the more recent contributors to this subject are Steindler,[9] Allison,[10] Leriche,[11] Key,[12] Sigridson,[13] Speed,[14] and others.

Anatomical Considerations.—The synovial membrane of the knee-joint is the largest in the body, and lines the capsule of the joint. Three arbitrary spaces are recognized (Fig. 1); the suprapatellar space found in front of the femur and beneath the extensor quadriceps tendon above the condiles forms a large pouch, which on distension with gas assumes the form of a gall-bladder. (Fig. 6.) The infrapatellar space is an extension of the synovial membrane into the centre of the joint, between the articular surface of the femur and tibia, and meets the semilunar cartilages, where it is reflected into their upper surfaces around their free inner margins, and back to the portion called the coronary ligament, which it lines down to its tibial attachment. The outline of this space, when filled with gas, is rectangular. The synovial membrane is reflected onto the crucial ligaments, which it invests except behind and below, and thus shuts them out of the synovial cavity. The infrapatellar space is divided by the pad of fat, a loose areolar connective-tissue structure, more or less triangular in shape. The base is directed downward and forward, and its apex is directed to the centre of the joint. This pad is composed of fatty and loose fibrous tissue and is invested by synovial membrane. From its borders extend small villus tags, covered by irregularly placed fibroblastic cells. The pad is held in position by well-defined ligaments, known as the alar ligaments. These ligaments are extensions, or reflections of the synovial membrane, and are found on the lateral peripheral margins of the fat pad, and extend upwards, adapting themselves to the outer and under surface of the patella, blending with the capsule on the under surface of the quadriceps extensor tendon. From the apex of the pad of fat a fold extends to the anterior surface of the

crucial ligament and is called the ligamentum mucosum. The pad and its villi are very vascular. Their function is that of wiping the joint surfaces with each movement of the knee. In inflammations, and infections of the joint, the pad becomes hyperplastic, and the villi are elongated, and thickened, often filling the infrapatellar space.

The synovial membrane, which plays the most important part in inflammatory processes of joints, does not cover the articular cartilages of the bones which enter into

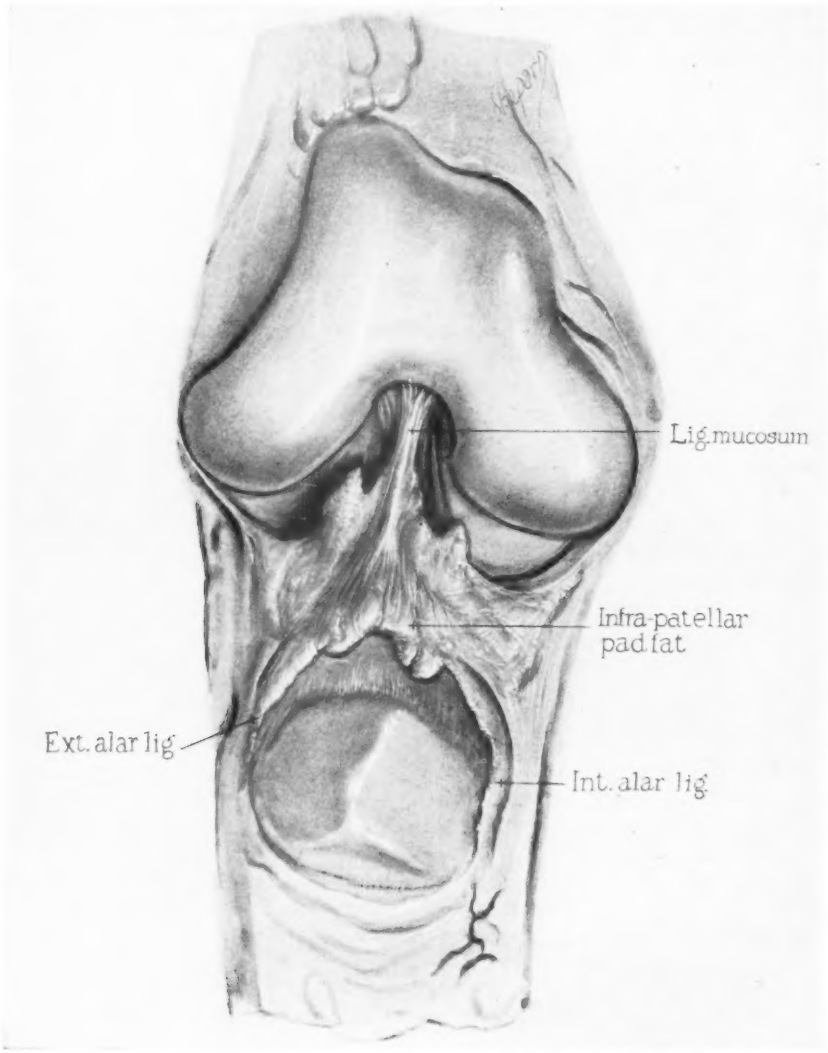

FIG. 1.—Shows the infrapatellar pad fat, and its villus tags. The articular cartilage of the femur is not covered by synovial membrane. Note its distribution and attachments above the condyles.

the formation of the joint, but extends to the edges of the articular cartilage. The capsule of the joint consists of two layers, the stratum synoviale, and the stratum fibrosum. The lining of the joint capsule constitutes the stratum synoviale. The surface of the stratum synoviale is covered by a layer of mesenchymal cells forming no definite lining membrane. It is believed by many histologists that the surface cells resemble true mesothelium, or endothelium, but these cells are now considered to be a modified fibro-

blastic cell, arranged loosely and irregularly to form a continuous smooth surface layer. There is, however, no true epithelium, endothelium, or mesothelium covering in synovial membrane surfaces, that is, the cell limits are not demonstrated by silver nitrate. Hence, the term synovial mesothelium, epithelium, or endothelium, should perhaps be interpreted as meaning areas in which the cells of the synovial membrane surface are closely packed and thus resemble endothelium, or mesothelium. Hueter,[15] in 1866, employing the silver impregnation of Von Recklinghausen,[16] caused considerable controversy when he denied the existence of epithelium lining synovial serous surfaces. This view was later confirmed by two independent workers, Hammar[17] and Braun,[18] who, in 1894, published their result, coming to practically the same conclusion as Hueter.

Arey[19] states that the joint lining in the embryo appears as an undifferentiated mesenchyman homogeneous substance. A cleft appears between the two bone surfaces, and cells from the mesenchymal substance flatten out and line the cleft. The cell limits determine the joint cavity and its synovial surface. The cells in a joint are not so far advanced in their development as are the cells in other cavities, which line serous surfaces. They, therefore, are not as definitely differentiated as are those of other cavities, such as pleura, or peritoneum. The cells, however, resemble endothelium in their structure and in their function. Jordan[20] states that the surface of synovial membranes is covered by a single layer of mesenchymal epithelium. Lewis and Stohr[21] also speak of these cells as being mesenchymal epithelium. Key states that the character of the cells found in fluid of normal joints is further evidence that joint cavities are clefts in the connective tissue, which are incompletely lined by slightly modified connective tissue.

The cellular elements do not form a complete mosaic covering over the surface of the synovial membrane. Dense connective tissue fills in the spaces so as to form a uniform and smooth gliding surface. The connective-tissue stratum varies in density in different locations of the joint. In places where the synovial membrane participates in an active gliding mechanism the loose fibrous tissue is quite abundant, but where motion is not so active the fibrous tissue is more dense.

While it may appear that undue space is devoted to a description of the cellular structure of the synovial membrane, in a paper such as this, dealing with a clinical problem, this phase is of interest from the standpoint of tissue regeneration after synovectomy. The question which is most frequently asked is, "What occurs to the joint lining after it has been surgically removed?" It has been found clinically by Allison, and shown experimentally by Key and Wolcott[22] that the synovial membrane reforms within a period of ninety days after its complete removal. If the synovial membrane is of epithelial or endothelial nature, it would follow that epithelization of such a large denuded surface would be rather difficult, unless enough tissue were left. This is not the case in the operation of synovectomy, for a considerable extirpation is done, leaving a large, raw surface. However, if the surface cells are fibroblastic, regeneration is more likely to occur. The synovial membrane reforms within a period of about ninety days, and can be "distinguished with difficulty from the normal."

It would appear that the question is still unsettled as to the nature, structure, and manner of regeneration of synovial tissue. Whether the synovial membrane regenerates or is replaced by fibrous tissue, which as a result of use becomes specialized and assumes all the characteristics of synovial membrane, even to the degree of secreting synovial fluid, is a question for

physiologists and histologists to determine. Suffice it to say that the histological controversies do not influence the clinical side of the question.

Secretory and Absorptive Power of Synovial Membrane.—The older anatomists and physiologists maintained that the synovial membrane secreted a mucigenous material, like the white of an egg, for the purpose of lubrication of joints. They further stated that this material was secreted by the endothelial cells of the synovial membrane. Controversy exists regarding the origin of synovial fluid, thus Fisher[23] believes that mucin enters the joint from the surface cells, from the deeper cells which empty the synovia through the villi, and also from the detached surface cells which have undergone a mucigenous degeneration. Allison[24] and his co-worker found an analogy between synovia and plasma. They found a variation in the protein, chloride and sugar content in arthritic synovia. Key[25] states that the cells of the macrophage series are the most important cellular constituents of normal synovial fluid. The origin of synovial fluid becomes still more confusing when the conception of the secretory function of the surface cells is debated. It was very simple to explain the origin of synovia when the surface cells were believed to be endothelial in nature, and the synovia the product of endothelial-cell secretion, namely, mucin. But when the surface cells are classed as fibroblastic in character, the origin of synovia must be looked upon as coming from a different source. Effusion into the knee-joint as a result of irritation from trauma or infection is quite marked. When the knee-joint is injected with a 1 per cent. acuous iodine solution, it becomes promptly distended by a serous exudate. I have also injected dialyzable and soluble materials into the veins of dogs, but they were not readily recoverable from the joint synovial fluid. We injected iodides or methylene blue into the veins of dogs, and the material was readily recoverable from the saliva and the urine, but it was not found in the joints by the usual tests. Injection into the joint synovial surface is promptly absorbed and is found within a short time in the neighboring lymph-nodes.

Noetzel[26] injected *Bacillus pyocyaneus* into the knee-joints of rabbits and found that from five to ten minutes later the organisms were present both in the inguinal lumbar and crural lymph-nodes, and in the circulating blood. Pawlowsky[27] has demonstrated the presence of staphylococci in the blood, and in organs of animals from twenty-four to forty-eight hours after inoculation of the knee-joint. He has shown that this dissemination is inhibited or wholly prevented, if before inoculation, an acute inflammation of the joint has been produced by the injection of some sterile irritant. Opie[28] states that the lymph-nodes during the first hour after inoculation are not efficient filters for bacteria. While the quiescent lymph-node is an inefficient filter, the inflamed node containing even at an early period of infection many bacteria, is effective in restraining the dissemination of bacteria into the general circulation. He further states[29] that a great variety of substances which are either non-dialyzable, or insoluble in water are dissolved and removed when introduced into the tissues of an animal. India ink, Berlin blue, and trypon blue were injected into the knee-joints of dogs by Key,[12] Sigridson,[13] and Rynearson[30] to demonstrate absorption and dissemination by way of the regional lymph-nodes. The process of removal from the synovial membrane is by means of the macrophages.

It can readily be seen that the products of decomposition necrosis, or infection of the synovial membrane of the knee-joint, can be taken up by phagocytic action, carried to the regional lymph-nodes, and into the general circulation. The absorption from the joint varies with the type of material it contains, and the state of the synovial membrane. In acute septic arthritis the absorption and dissemination are rapid; in chronic arthritis the absorption depends upon areas of healthy tissue.

Pathology.—The synovial membrane plays the most important part in inflammatory processes of joints. Upon entering an arthritic joint, one finds an increase of synovial fluid. In chronic arthritis the amount of fluid varies

with the pathological process and depends upon exacerbation or recession of the inflammation. It is usually considerable, and grayish-yellow in color, and often serosanguineous. It contains particles of detached granulation tissue, and plaques of gray mucus. These mucigenous plaques are found in the joint, floating in the synovial fluid, and frequently adhering to the synovial membrane, filling the recesses of the joint. Ashcroft, Cunningham, McMurry and Pemberton,[31] in a study of fifty cases of arthritis, found one positive culture in the synovial fluid, all of the others being obtained from the synovial membrane and articular cartilage and bone.

The synovial lining of the joint is very much increased in thickness. The fat pad is hyperplastic, thick, elongated, and succulent. The villi are increased in number and size, often filling the joint space. The articular

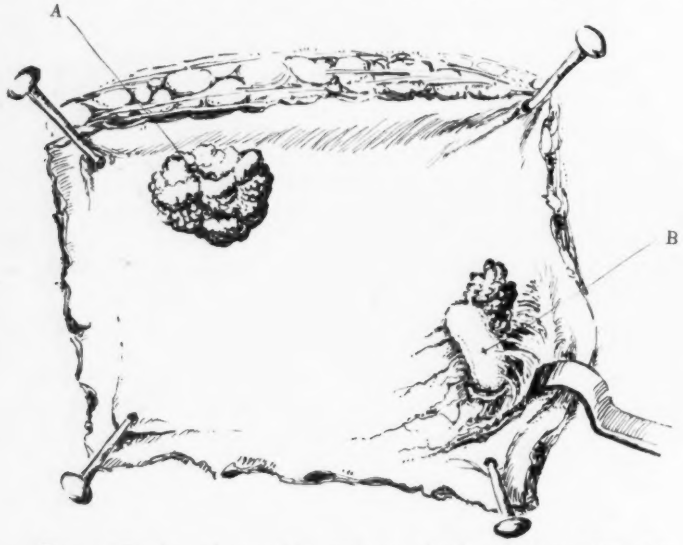

FIG. 2.—Portion of synovial membrane showing (A) cauliflower-like calcified area with granulation tissue surrounding it. (B) Large wart-like calcified process, with pannus on its upper border. Marked granulation tissue and fibrous bands holding it firmly to the synovial membrane.

cartilage appears pearly gray, smooth, with no erosions. Erosion and destruction of the cartilage occur in the adhesive form of arthritis of the polyarticular type. *In this form, synovectomy should not be performed.* In certain forms of arthritis the articular cartilage shows spicules of proliferated bone at the margins of the synovial attachment, but the gliding surface, the surfaces of contact and pressure, are rarely eroded. Bands of fibrous tissue may be present; these extend from the synovial surface, and stretch across the joint spaces. These bands are more prevalent in the suprapatellar space. Pannus formation is a common finding, and is more frequently seen over the femoral condyles. Calcified material may be found in pouches created by the hyperplasia of the synovial membrane, or attached to the tips of villus processes. (Fig. 2.) True cartilage is rarely found. Cartilaginous bodies are a clinical entity of an entirely different nature, and are associated with osteocartilaginamatosis. However, in only three cases have I found carti-

laginous bodies, and these were associated with disturbances of the semilunar cartilages.

Microscopically.—The most striking picture is that of new vascularization round-cell infiltration and cell proliferation. (Fig. 3.) The lymphocytic infiltration is of the subsynovial fibrous tissue. The peripheral cells proliferate so that several layers are deposited on the surface. Portions of the synovial membrane show a fibrous stroma with numerous new-formed vessels, around which are many lymphocytes. The cells appear in nests or clumps. Areas of normal synovial tissue are seen which suddenly disappear and the surface is covered by various sized excrescences of compact fibrous tissue, which in places shows a tendency towards hyalinization and fatty degeneration. True cartilages are rarely seen, but areas of calcification may be noted. (Figs. 4.) The areas in which polymorphonuclear leucocytic infiltration and fresh blood-cells are scattered in the tissue are indicative of an exacerbation of the infectious or irritative process. Plasma-cells of the polyblastic variety and wandering macrophages are evidences of chronic inflammation. Verruca may be distinctly papillary in character so that large excres-

FIG. 3.

FIG. 4.

FIG. 3.—Marked inflammatory changes with newly formed connective tissue and blood-vessels.
FIG. 4.—Marked thickening and fibrosis of synovial surface. Fibroblastic tissue in sub-synovial surface. New-formed blood-vessels filled with blood-cells. Fibroblastic proliferation of the villi.

cences are joined by thin pedicles of the synovial sheath. The vessel walls are increased in thickness, and their contents are full of red blood-cells and cells in a stage of degeneration. Decomposed blood pigment may be found within the capillaries or in the tissue. Bick[32] states that in a study of synovial membrane in chronic arthritis regardless of the eventual outcome of the studies in their etiology, the observed effect in the synovial tissue is within a reasonable range always the same.

In this series both the hyperplastic or atrophic, and the hypertrophic or osteo-arthritic types were operated upon. Changes in the synovial membrane of the two types differed slightly. In the hyperplastic type there was more dense fibrous-tissue formation, with perhaps a more marked round-cell infiltration. In the osteo-arthritic type there is a greater fatty and hyalin degeneration. It is often difficult to judge from the pathology of the synovial membrane alone what type of arthritis one is dealing with. Osteophytic deposits on the periphery of the articular cartilage may be noted in both. Synovectomy in this series was performed on several cases of recurrent

hydrops of the knee-joint (acute recurrent serous synovitis). One case was of interest because it showed a recent strangulation of a villus with thrombosis of its vessels. (Fig. 5.)

Indications for Operation and Results.—No fast rules can be established for the performance of this operation. Cases should be selected with the utmost car. In general, any arthritic joint which is associated with pain, swelling, limitation to the normal range of motion, and X-ray indications of clouding and thickening of joint spaces should be subjected to the operation of synovectomy.

Often the pain is general over the knee-joint, but as a rule the patient will indicate a more definite point, on the inner side of the knee below the

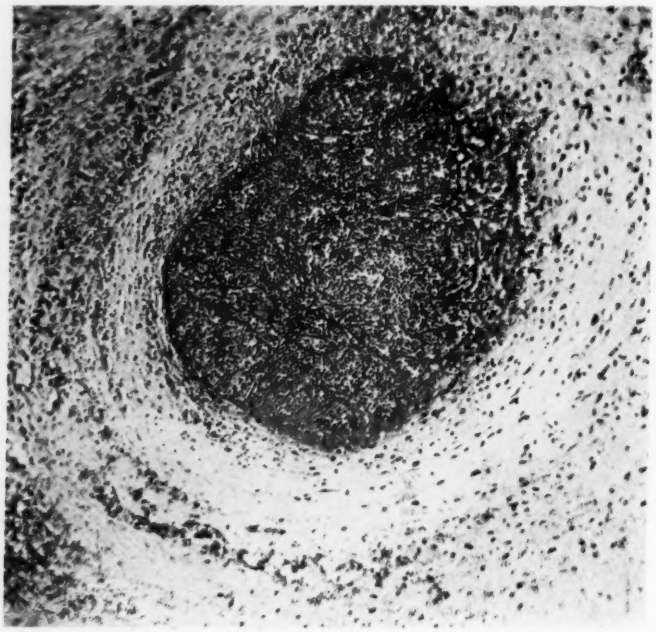

FIG. 5.—Organizing thrombus. Note the inflammatory cells surrounding the vessel.

lower border of the patella. The reason for this is the greater amount of strain put upon the inner side of the knee due to weight-bearing. There may also be pain in the popliteal region. This is due to tension in the posterior compartment of the knee-joint as a result of accumulation of fluid and to a shortening of the ham-string muscles and to a contraction of the posterior capsule. Occasional complaint is made of pain at the head of the fibula. This is caused by the involvement of a branch of the peroneal nerve.

Effusion in the joint is very common. The swelling may be uniform, causing a prominence above the patella. More often there are irregular indurated masses below the patella, and on the lateral surfaces of the joint.

Limitation of motion is almost invariably present. Both flexion and extension of the joint are affected, giving rise to faulty weight-bearing.

The röntgenogram shows a thickening and clouding of the spaces in the joint which are normally found clear. This obscurity is due to synovial thickening, and to the accumulation of fluid. The method employed in our earlier cases to visualize these joint spaces, was by the introduction of carbon dioxide. (Fig. 6.) By this means the outline of the synovial membrane, its distortions and any foreign substance otherwise invisible could be easily determined.[33] Changes on the periphery of the articular cartilage such as

FIG. 6.—Lateral view of knee-joint inflated with carbon-dioxide gas showing enlargement of pad of fat, distension of the suprapatellar space, and bands of adhesions running through it. The posterior compartment of the knee-joint is clear. This patient had a hypertrophic villus synovitis.

hypertrophic deposits are no contra-indications for operation. On the contrary, they confirm the pathological picture which may be expected in the joint. Nor is age a determinative factor in operation. The youngest patient I have operated on was twenty-five, and the oldest sixty, the average age being forty. Of twenty-five patients operated on, fifteen were men. Of these cases, twenty-two had good results, one was a poor result, one developed

an ankylosis, and one had a recurrence of pain about six months after the operation. Of the twenty-two cases, 50 per cent. had completely restored function, complete loss of pain and no recurrence of swelling. Of the others, the results varied with the general physical condition of the patient, and in accordance with the other joints involved. In the cases of multiple arthritis, the other joints were distinctly benefited by the operation. It was surprising to note the increase of circulation to the extremities, and the added sense of warmth and well being. The function in the joints improved with the lapse of time, so that the oldest case, a woman sixty-five years of age, who was operated on in 1925, has a complete range of motion and freedom from pain. One must not judge the results of synovectomy the first few months after the operation. Improvement continues with the use of the knee.

Of the three bad results, one developed a secondary infection after the operation, which resulted in ankylosis. One case was a multiple hyperplastic arthritis with erosion of the articular cartilage, and resulted in a limitation of motion. The range of motion obtained after the operation was not less than the patient had before, but nevertheless it is counted as a bad result. The third patient was also a multiple hyperplastic type and had a recurrence of pain soon after the operation. It may be stated that when there is an erosion of the articular cartilage, the results are not apt to be good as to the complete return of function, but improvement of the other joints may occur. It may also be stated that the male patients without exception had excellent results, the joints returning to comparatively normal state as to pain, range of motion and weight-bearing. The pain and discomfort after synovectomy are no greater, and in my opinion are less than after the removal of a semilunar cartilage, and the time of disability is not longer. In no case was there a complication as a result of the split patellar approach to the joint.

Prognosis.—First, there is the ever-present danger of infection. This can, however, be obviated if not completely eliminated by a careful observation of Lane technic, and the observance of asepsis to a superlative degree. It must be remembered that due to the chronic irritation in the joint, the synovial membrane has become thickened and the lymph-nodes blocked; therefore, secondary infection of the joint is not followed as frequently by systemic absorption. In normal joints infection is followed by a rapid absorption and dissemination by the unprepared lymphatics. This warding-off process Murphy[32] called "Cofferdamming." Although the danger of systemic infection is minimized, the effects upon the joint are nevertheless associated with danger of ankylosis.

Secondly, there is the danger of injury to structures upon which the stability of the joint depends, such as the crucial and lateral ligaments.[35] This may be easily avoided by finding the line of cleavage of the lining of the joint, and not penetrating too deeply into the peri-articular structures.

Thirdly, hæmorrhage may seem alarming coming from a large denuded surface with no evident vessels for hemostasis. Compression, however, con-

trols this bleeding and the hemoarthrosis that follows need not be aspirated since it is absorbed without difficulty.

Operation.—The anæsthetic we have used on our last cases has been the intraspinal injection of spinocaine. This has proved very satisfactory, especially in those cases associated with cardiac complications.

The knee-joint is prepared the night before the operation by shaving, thorough scrubbing and antisepsis. The patient is placed in a recumbent position on the operating table with the knee slightly flexed. A tourniquet is placed around the thigh. The knee is again prepared by careful antisepsis, iodized and made ready for operation.

The incision is one originated by me[34] and is now quite generally used. (Fig. 7.) From a point about two and one-half to three inches above the superior border of the patella in a mid-line the incision is continued downwards to the upper border of the patella, circling the outer border, to the lower mid-line, and ending at the tibial tubercle. The skin is then reflected on both sides, exposing the tendinous portion of the quadriceps extensor, and its covering over the patella. A linear incision is now made in the mid-line through its full thickness. By means of a chisel the patella is split. This leads directly into the joint. Scissors are introduced through this opening and the incision is carried upward and downward through all the structures. By this means the infrapatellar pad of fat is cut in two. As a rule a considerable quantity of fluid and blood escapes from the joint, and with it a grayish mass of mucus. This mucigenous substance is very tenacious and is often adherent to the synovial surface, especially to the suprapatellar space. This by-product is entirely wiped out by means of a sponge, leaving a dark and reddish hyperæmic and thickened synovial membrane exposed.

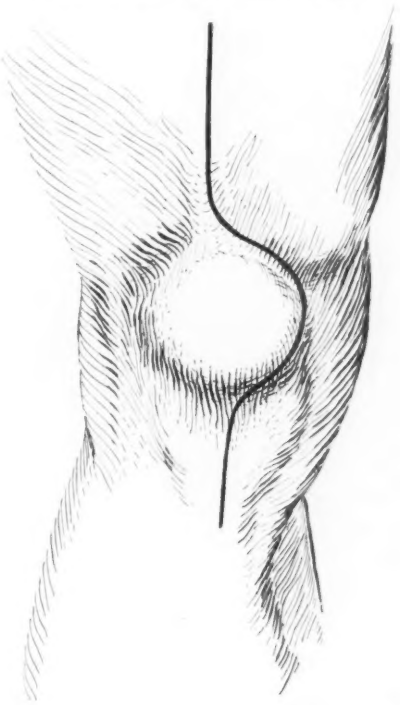

FIG. 7.—Incision for approach to the knee-joint, first described by the author and has been used where a wide approach is desired. This incision has the advantage in avoiding a scar over the patella.

The knee is now flexed to its full limit. The split patella is retracted, and the dissection and extirpation of the synovial membrane are now begun. By means of scissors and tissue forceps the split pad of fat is now dissected out (Fig. 8), first one side and then the other, and with it as much of the synovial membrane on the lateral joint surfaces as is possible, avoiding injury to the semilunar cartilages. Some operators advise the removal of the cartilages, notably Allison.[10] When there is erosion, it would seem advisable to remove them. However, I have never found occasion to do so.

The synovial membrane from the suprapatellar pouch is now dissected out, which completes the synovectomy. Before closure a search should be made for any loose tags of synovial membrane so as to leave the joint with a smooth and clean surface. All hæmorrhage is controlled by either ligation or hot packs. The closure is made by sewing the capsule from above downward by continuous interlocking stitch, down to the upper border of the patella. Interrupted sutures are then introduced through all the

layers, covering the patella, paying special attention to coaptation of the bone edges. The lower remaining portion of the incision is then closed in the same way as is the upper. Chromic catgut is used for all deep sutures. The subcutaneous tissues are closed by a running plain catgut suture, and the skin incision is closed by means of dermal horsehair or silk.

No drain is ever used in the joints. A posterior molded plaster splint is applied. This is retained for about six days, when it is removed and passive motion executed. The patients are allowed out of bed in a wheel-chair about the tenth day, and encouraged to get around on crutches as soon as the pain subsides. Active and passive exercises are carried out by a trained physiotherapist, and weight-bearing allowed at the end of the second week.

The dressings are not disturbed for ten days, and the sutures not removed until the skin edges show signs of healing. Too early removal of the sutures often leads to a retraction of the incision, resulting in secondary infection.

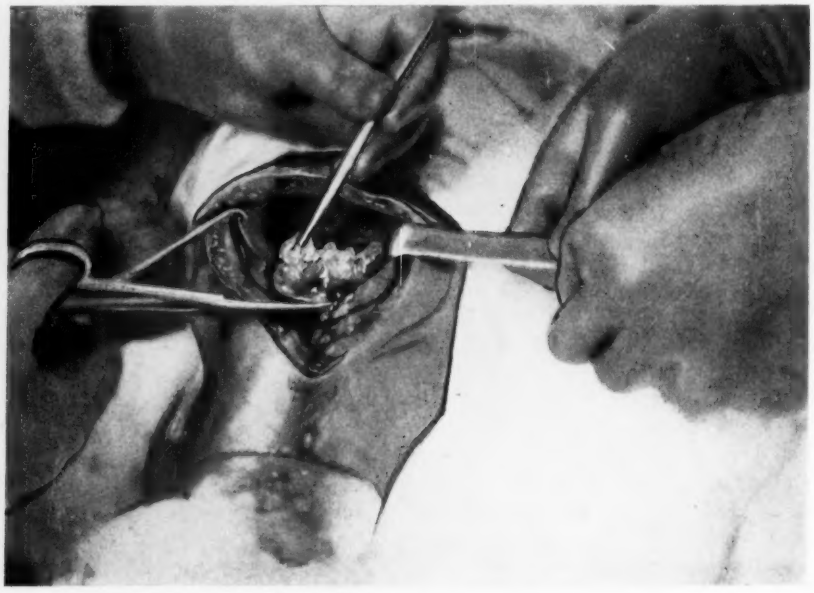

FIG. 8.—Dissection of fat pad and synovial membrane.

If effusion or distension of the joint occurs as a result of an oozing hæmorrhage into the joint, it is seldom advisable to aspirate, since absorption of the fluid or blood takes place within a short time after the operation. There is always a danger of secondary infection from aspiration. This results by introduction of microörganisms through the puncture, which leaves a puncture wound, allowing a constant oozing of serous and serosanguineous fluid.

The application of heat by means of infra-red lamp lessens the pain after the operation. The dressings must not be disturbed when heat is applied. A few grains of codeine help to control the pain.

Faradic current applied to the muscles of the leg and thigh helps to maintain muscle tone, and restores function more quickly. This is especially true of the quadriceps group.

The degree of pain is surprisingly little in proportion to the magnitude of the operation. Patients have commented that the pain in the joint before operation was greater than the pain from the operation. Most of the patients stated that the pain and discomfort in the other joints were definitely improved. This observation has also been made by other surgeons. Patients who were suffering with a bilateral arthritis of the

knees refused operation on the second knee because the pain in the unoperated knee was mitigated as a result of the synovectomy.

Conclusions.—(1) Synovectomy in properly selected cases, and in experienced hands, gives rise to striking results.

(2) The operation can be easily performed under spinal anæsthesia.

(3) In most cases it lessens or removes pain, improves the range of motion in the joint, and gives rise to a warmth and sense of well-being in the extremity.

(4) There is an improvement in other joints in cases of multiple arthritis.

(5) The improvement of function continues long after the operation.

(6) The age of the patient is no contra-indication.

(7) The adhesive or the multiple proliferative types with erosion of the articular cartilage give the poorest results.

(8) The monarticular osteo-arthritic, and the atrophic without erosion of the articular cartilage give the best results.

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SITUS TRANSVERSUS VISCERUM

REPORT OF CASE WITH CHOLELITHIASIS

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The primary interest of situs transversus, situs inversus viscerum, or transposition of the thoracic and abdominal viscera, is merely academic, as its incidence is a medical curiosity. Existence of this anomaly is entirely compatible with normal health, longevity, and reproductivity. Major interest, however, attaches to its occurrence in individuals with disease conditions requiring surgical relief.

Estimations of incidence are at variance. Upwards of 400 cases have been reported, but this by no means furnishes a true record of its incidence; undoubtedly many more cases have occurred than have been reported, because they have escaped anything more than casual notice.

It has been reported almost entirely in connection with the so-called left-sided appendicitis. But the majority of these case reports leave a great deal to be desired in the way of data sufficiently conclusive to classify the cases as bona fide transposition, failure of colon to retate, or simple wandering appendix due to mobile cæcum, long mesentery, adhesions, *etc.*

The most recent and reliable figures I have been able to find are those quoted by Counseller, who states: "In The Mayo Clinic, ten cases were observed in a registration of 347,000 patients between 1910 and 1927. . . . It was noted once in 35,000 routine examinations in the army." The remarkable parallelism of these figures is very striking.

In the presence of true dextrocardia one is probably justified in assuming that the abdominal viscera as well are transposed, as the condition is generally complete. However, comprehensive skiagraphical examination of the gastro-intestinal tract is the only scientific method of determining the question. And in the final analysis, only operative or necropsy findings are conclusive.

In the individual whose case report is abstracted below, the transposition was complete. There was dextrocardia; the larger lobe of the liver was on the left and carried with it the gall-bladder, which emptied into the duodenum to the right; the pylorus pointed to the left; the cæcum and appendix were in the left iliac fossa, and the sigmoid occupied the right iliac fossa.

Case.—A woman, aged forty years, white, entered St. Joseph's Hospital August 14, 1931, for interval cholecystectomy and appendectomy. The diagnoses on admission were: (1) Cholecystitis, chronic, non-suppurative, moderately severe, with cholelithiasis, the gall-bladder skiagraph exhibiting multiple faceted calculi. (2) Situs transversus viscerum, complete. (3) Arthritis, chronic, recurring, non-suppurative, multiple, proliferative type, involving all the joints, large and small, of both extremities, and the

cervical and upper half of the dorsal spine; characterized by progressive limitation of motion and function with deformity of involved joints, most marked in wrists and smaller joints of both hands. (4) Pyelitis, chronic, suppurative, mild, bilateral. Insofar as the transposition of viscera is concerned, the early history of the patient was of importance only secondarily, as showing the many vicissitudes of disease through which the patient had passed without the anomaly having been discovered. She had experienced the usual diseases of childhood and at the age of ten years had an attack of acute rheumatic fever which involved practically all the larger joints successively. However, she made a good recovery without residual effects.

She stated that from childhood she had always been conscious that the maximum heart impulse was on the right side, but that not till much later in life was the true significance of the anomaly discovered. She had never been told of the existence of any abnormality of her viscera until her present hospitalization.

At the age of twenty, while in training as a nurse, she was operated upon through the classical incision for right-sided iliac pain which was diagnosed as acute appendicitis. No appendix was found and patient states that she was told that the many adhesions covering the appendix made its removal inadvisable. Later, during the same illness, it was found that the patient was heavily infested with malarial parasites.

During the World War she entered the military service as a nurse and served in hospitals along the northern Atlantic seaboard. She contracted influenza in 1918, and about one month later developed an acute arthritis which localized chiefly in the ankles, shoulders and hands. She did practically no duty for four months, though apparently not ill enough to be hospitalized. She was then transferred to a milder climate and was able to do duty for about only two months before she was hospitalized May, 1919, with a diagnosis of chronic fibrinous pleurisy of both bases; this condition was complicated by subacute arthritis of all joints of both extremities and both mandibles. There was only slight improvement in hospital, and she was discharged from the military service three months after admission, on certificate of disability. No mention was made of the situs transversus in her medical records on admission or discharge from the military service.

Since 1919, the patient has never been entirely free of joint symptoms; the arthritic process has manifested itself by periods of remissions and exacerbations at varying intervals. There has been a steady progression in the disability due to limitation of motion and function in the involved joints. Though there does not appear to be bony ankylosis as yet, extra-articular fibrosis, contractures, and trophic changes have resulted in crippling and unsightly deformities, particularly of the hands.

During the past few years the patient has been treated at intervals for cystitis and pyelitis of low grade. It was during one of the cystoscopic examinations that transposition of the viscera was discovered by the urologist.

The patient was operated upon under spinocain, synergized by intravenous sodium amytal: the anæsthesia was ideal. Aside from the difficulty of disposing the limbs of the patient on the table, because of the arthritic deformities, the operation was absurdly easy. The entire anatomy of the abdomen was reversed. The gall-bladder with contained stones and a fat appendix were removed through a left rectus incision.

The patient made an uninterrupted recovery and left the hospital twenty-three days after operation. When sufficient time has elapsed to assess the benefit derived from the removal of these possible foci, it is contemplated to attempt rehabilitation of the patient's joints by plastic surgery. The possible application of sympathectomy has been considered.

Explanation of the origin of this condition must be largely speculative. There are certain things we know and others we may surmise. (Fig. 1.) For instance, the anomaly is teratological without doubt, and occurs at an early stage of phylogenetic differentiation of the embryo. Observation of

this mirror transposition in duplicate monsters has led to the suggestion that the person exhibiting it is the right half of an original duplicate monster in which the left was resorbed. However, the idea is not tenable because the anomaly is rare in one-egg or identical twins; and the latter are merely a variation and the usual one, of bisomatous monsters. Moreover, there is no embryological or logical reason why the simple fact of division of the blastomere *per se* should be followed by abnormal development in a particular half, either the right or left.

FIG. 1.—At first glance one would think of kidney stones. Second thought brings doubt, chiefly because of the structure of the stones.

Embryology furnishes the most acceptable explanation in the development of the cardiovascular system from the primitive aortæ and aortic arches. The sites of the abdominal viscera are determined by the course along which their circulation evolves. In the normal schema, the arch of the aorta is formed by the fourth left aortic arch; the descending aorta by the fusion of the right with the left dorsal aorta; the fourth right arch and the right dorsal aorta disappear caudad of the third arch. If this process is reversed, the fourth right arch persists and forms the aortic arch; the heart occurs on

the dexter side; the aorta arches to the right and the descending aorta and subsequent venous system development bring about the sinister position of the abdominal viscera.

Analogous confirmation of this is obtained by a study of comparative anatomy. In fish, the aortic arches persist and give off branches to the gills where the blood is oxygenated; in birds the fourth right aortic arch forms the arch of the aorta; in reptiles the fourth arch on both sides persists and there is a double aortic arch.

FIG. 2.—Skiagraph confirms dexter position of stomach and clinches diagnosis of situs transversus. Needless to say, complete röntgenographical examination of the entire gastro-intestinal tract was had.

It is most remarkable that this patient, who is herself a graduate nurse, could have passed through so many medical hands without the existence of this anomaly having been definitely established, until she was forty years of age. Dextrocardia was tentatively diagnosticated in 1919, during the course of a skiagraphic examination (Fig. 2) of the chest for symptoms suggestive of pleurisy. At this time complete transposition of all viscera was suspected and skiagraphic examination of the gastro-intestinal tract was requested by

the radiologist; there is no record, however, of its having been done. Neither was the patient advised of the significance of the above observation.

The physical examinations of this patient on admission and discharge from the military service do not mention any departure from the normal insofar as the heart is concerned, nor does it appear that the anomaly was enumerated as a diagnosis on her clinical records.

It is recalled that she was operated upon at twenty years of age for appendicitis; the usual right rectus incision was used and no appendix found. Obviously the underlying condition was not recognized. The right-sided pain had by the patient can be explained by splenic pain incident to a malarial infestation discovered subsequently.

The lesson to be learned from this case is *the fundamental importance of a careful physical examination of all patients.* It can be argued that transposition of viscera is an unlikely contingency: that beyond a needless and fruitless operation in the case at issue, no great harm has resulted from failure to recognize the anomaly. Still, it is hardly a matter for congratulation.

BRIEF COMMUNICATION

MORTALITY FOLLOWING GALL-BLADDER SURGERY

AMONG the many excellent articles on biliary surgery in the September, 1933, number of ANNALS OF SURGERY is one by Eiss, entitled "Conservation of Hepatic Function in Gall-bladder Operations." This article again draws our attention to a very important point, namely—the occasional sudden and unexplained death following operation on the biliary tract, which, for want of a better name, is called "liver death."

Eiss, in his communication, discusses the various tests of liver function that we now have at our command, mentioning in particular the dye-retention test as described by Graham, and shows that in the Barnes Hospital where this test is used routinely in all patients upon whom any type of biliary surgery is contemplated, the mortality of cholecystectomy has been reduced from 6 per cent. to .4 per cent. This reduction has been brought about by treating pre-operatively with glucose and calcium the patient with excessive dye retention.

We know that the liver has a multiplicity of functions and at the present time we have no definite test that will give us a true index of the amount of liver function present in the presence of biliary disease, although perhaps the dye-retention test is our best test. We do know that the metabolism of glucose is an important function of the liver and apparently the function most disturbed by infection in the liver and biliary tracts.

Until we have some fairly exact liver-function test available to surgeons who are compelled to operate upon patients with gall-bladder disease, comparable to the kidney-function test, it would seem that every patient before undergoing an operation on the gall-bladder or biliary passages should be looked upon as a bad risk and treated as such in more or less of a routine manner as described below.

The purpose of this communication is to point out that the mortality in gall-bladder surgery which is high can be reduced by looking upon every patient with disease of the biliary tract, whether jaundiced or not, as a bad risk and by giving adequate pre-operative treatment with glucose to every patient. If one is working in a clinic or has at his command the facilities to carry out the dye-retention test, perhaps it is safe to treat pre-operatively only the bad-risk case, but the bulk of gall-bladder surgery throughout the country is done by men who do not have facilities for carrying out this test. By regarding every patient as a bad-risk patient, the mortality certainly can be reduced.

Practically it is quite simple, both for the patient and the surgeon, to build up the glucose reserve before operation. In this day of economic stress where pre-operative hospitalization is a financial hardship in many instances, the

patient can be instructed to take sugar in large amounts for several days before going to the hospital. In the acute case which requires immediate hospitalization, the glucose can be given by mouth if well tolerated, and if not it can be given intravenously. By this simple procedure the so-called "liver death" can be reduced to a minimum.

In addition to increasing the glucose reserve of the patient, several other points are important and quite essential, and these are as follows:

(1) A carefully executed operation, paying due respect to the anatomy of the bile passages, *etc.*

(2) A carefully selected anæsthetic. In my own experience, spinal anæsthesia is the anæsthetic of choice in the patient who is potentially a bad risk. Avertin supplemented with ethylene is second choice, and ether is the last to be chosen and is seldom used in my own practice.

(3) Continuous gastric lavage by means of an indwelling nasal catheter if there is any tendency to regurgitation and dilatation of the stomach. Of course the fluid balance during the first few days must be maintained, and in case of vomiting this is done by giving glucose solution intravenously and subcutaneously.

By this simple and more or less empirical procedure of building up the glucose reserve in all patients about to undergo biliary surgery, a great many lives can be saved and the number of so-called "liver deaths" kept to a minimum.

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MEMOIRS

WILLIAM McDOWELL MASTIN

1853–1933

Dr. William McDowell Mastin was born in Mobile, Alabama, July 3, 1853. He was descended from a long line of ancestors, originally of English stock, who in the territorial days of Alabama had settled and prospered in

William McDowell Mastin, M.D.

Huntsville. A typical Southern gentleman by character and birth, Doctor Mastin inherited a pride in his medical lineage which was historical on both sides of his family tree. His father, Dr. Claudius Henry Mastin, was recog-

nized by the profession as the "Nestor of Surgery" in the South. He, in turn, had been influenced in his choice of a medical career by the example of his grand-uncle, Dr. Claudius Henry Levert, a celebrity in the early history of the state. Again, Doctor Claudius, the father, had married Miss Eliza McDowell, a lineal descendant of Ephraim McDowell, of Dansville, Kentucky —the "Father of Ovariotomy"—for whom young William was named. From this union four children were born, two sons and two daughters. The sons, following the example of the father who had graduated M.D. in the University of Pennsylvania in 1849, took to Medicine and were likewise graduated M.D.'s in the same university; the older, William McD., in 1874, and Claudius Henry, Jr., in 1884. The loyal attachment of the Mastins for the University of Pennsylvania was a marked characteristic of the family.

One cannot refer to Dr. William McD. Mastin's family antecedents without recalling the important rôle played by his distinguished father in the early history of the foundation and organization of the American Surgical Association. In fact, Dr. Claudius Mastin, a personal friend and devoted admirer of Dr. Samuel D. Gross, the illustrious founder, became from the foundation in 1880 one of Doctor Gross' most active lieutenants in the organization. Though an ardent Confederate and surgeon of high rank in the Southern army, Doctor Mastin was one of the first after the war to disregard all sectional prejudices and party feeling in uniting the medical profession of the North and South, and in restoring the national spirit in medical and scientific associations of the country. That his services were duly appreciated is shown by his successive election to the second vice-presidency in 1883–1884, the first vice-presidency in 1889–1890 and to the presidency in 1890–1891. Furthermore, he was one of the most influential members of the Council from 1891–1896.

It was through the untiring efforts of Dr. Claudius Mastin, with the coöperation of the Fellows of this association and the Alumni of Jefferson Medical College of Philadelphia, that the Samuel D. Gross monument in Washington was erected. He wrote the dedication on the monument and his address at the unveiling is a beautiful tribute to the illustrious founder and a model of chaste and eloquent speech.

Not content with his work in the American Surgical Association, Doctor Claudius founded and organized the Triennial Congress of American Physicians and Surgeons. He also shared in the founding of the American Association of Genito-Urinary Surgeons in 1886 (President in 1895–1896) and was one of the organizers and trustees of the First Pan-American Medical Congress in 1891.

These and other numerous activities in national and regional organizations are mentioned not only to recall Dr. Claudius Mastin's invaluable services to this (American Surgical) Association, but because of the influence that his commanding position exercised upon the character and future career of his oldest son, William, who also became a Fellow of the Association in 1887.

William McDowell Mastin was graduated as a Doctor of Medicine at the medical school of the University of Pennsylvania in 1874, just as his father had been twenty-five years before him. He supplemented his medical course by an interneship of a year in the University Hospital and another in the Wills Ophthalmic Hospital of Philadelphia. Then followed a tour devoted to the medical clinics of London and Paris. He returned to Mobile in 1877, where he settled permanently to practice medicine in association with his father. In 1898 Dr. Claudius H. Mastin passed away and his son, William, then forty-seven years of age, not only had succeeded him in his large practice but had become the leading surgeon in Mobile and was nationally recognized as one of the ablest and most scholarly surgeons of the South.

The years 1879–1913, that is, from his twenty-fifth to his fiftieth years, mark the period of Dr. William Mastin's greatest surgical and literary activity.

His earliest papers on "Ametropia in Its Relations to the Inflammatory Lesions of the Eye and Lids" (1879) and on the "Initial Lesion of Syphilis on the Palpebral Conjunctiva" (1880), show the influence of his early ophthalmic training.

Among his more massive and monographic papers are the following: A collective study on "Tracheotomy for Croup in the United States: an Analysis of 863 Operations" (Gaillard's Med. Jour., vol. xxix, 35 pp., N. Y., 1880); "Venous Blood Tumors of the Cranium, Connected with the Sinuses of the Dura Mater" (ANNALS OF SURGERY, 40 pp., 1885; enlarged in Jour. Am. Med. Assn., 93 pp., September, 1886); chapters on "Abdominal Section," Buck's Reference Handbook; "Ulcer and Ulceration," in Bryant and Buck's American Surgery, vol. iii, 1907; "Recurrence at a Late Period after Operation for Cancer of the Breast," based on personal observations (ANNALS OF SURGERY, vol. xlviii, 1908); "Résumé of the Surgical Treatment of Ano-rectal Imperforation in the New-born, with Report of Personal Cases" (Surg., Gynec., and Obst., vol. vii, 1906).

What may be regarded as his magnum opus and literary monument is his "History of Filaria Sanguinis Hominis; Its Discovery in the United States," based upon his personal studies of the parasite as he had identified in Mobile in a case of chylocele of the Tunica Vaginalis Testis, (ANNALS OF SURGERY, vol. viii, pp. 321–362, 1888) with a bibliography of 126 titles.

How highly he was esteemed by his colleagues in the South is shown by his unanimous election to Fellowship in the Southern Surgical Association in 1890, without the formality of an application—an unprecedented and unique compliment in that organization.

During the most active years of his career he was chief of the surgical staff of the Providence Infirmary and of the City Hospital of Mobile. It was by his operations in these hospitals and especially in the Providence Infirmary that his widespread reputation as an exceptionally skillful and conscientious surgeon was established.

At the age of seventy-five years he virtually retired, though still advising some of his old clients and faithful friends. In the meantime, his sight had been impaired and this added further to his seclusion by depriving him of the pleasures of his library, which had always been one of the greatest enjoyments of his studious and intellectual life. Finally, and just as he was

stepping on the threshold of his eightieth year, the end came in an attack of bronchopneumonia which peacefully and painlessly ushered him into the eternal slumber, February 3, 1933.

In November, 1882, Doctor Mastin married Miss Margaret L. Crawford, of Mobile, who survives him. Three children were born from this union: Margaret McD. and Claudius Henry, who died at an early age from diphtheria contracted before the days of antitoxin; and a daughter, Miss Zemula Crawford Mastin, now residing in Mobile, who lived to be, with her mother, the greatest comfort of his life.

RUDOLPH MATAS

WALTER ELLIS SISTRUNK

1880–1933

THE death of Walter Ellis Sistrunk is a loss to American surgery, to the American Surgical Association and to the community in which he carried on his work. His surgical career was outstanding because of a well-endowed mind, a thorough fundamental training, a long apprenticeship, an originality

WALTER ELLIS SISTRUNK, M.D.

of thought, tireless industry, and love of his work. Those who were closely associated with him in the early days predicted the high place he had attained in the surgical profession, and his death will be mourned not only by surgeons of this country but also by foreign surgeons who have on many occasions

paid tribute to the contributions through which he made conspicuous advances in many surgical fields.

Doctor Sistrunk was born in Tallahassee, Alabama, in 1880. He received the degree of Ph.G. in 1900, from the Alabama Polytechnic Institute, and of M.D. from Tulane University in 1906. He was interne in the Charity Hospital, New Orleans, from 1904 to 1906, was assistant house surgeon in the New Orleans Sanitarium from 1907 to 1909, and practiced at New Orleans from 1906 to 1909, and at Lake Charles, Louisiana, from 1909 to 1910. In 1911, he went to The Mayo Clinic as assistant in pathology and was appointed first assistant in surgery in 1912, assistant surgeon in 1914, and attending surgeon and head of a section in the division of surgery in 1915. From 1918 to 1929 he was associate professor of surgery, The Mayo Foundation, Graduate School, University of Minnesota.

During this period he displayed an intense interest in all surgical problems but particularly he had a large part in a study of the diseases of the thyroid gland, the breast and the colon and the development of surgical treatment of these diseases. The operation which he proposed for the cure of thyroglossal duct cyst revolutionized the surgical treatment of this condition. He was one of the early advocates in this country of the Kondoleon operation for elephantiasis. His surgical experience was enormous, and the thoroughness with which he assembled the results of this experience and the clarity with which he presented it gave to his publications both the weight of authority and unusually instructive qualities. For twenty years, moreover, parasitologists and internists have acknowledged a debt to him for pointing out that amœbiasis is widespread in this country.

The accidental and tragic death of Doctor Sistrunk's son, David, and his love for his homeland in the South led him to sever his connections with the clinic in 1929. He associated himself with one of his former assistants, Dr. G. D. Mahon, in Dallas, Texas, and here he continued to practice his profession until his death three years later. Those of us who knew him best will remember him for his kindliness, for his devotion to the highest surgical ideals and practice, for his enthusiasm, for his ingenuity and for his loyalty to his colleagues. These qualities, the devotion of his pupils, and the permanency of his scientific contributions will perpetuate the name and accomplishments of Walter Sistrunk.

DONALD C. BALFOUR

THE JUBILEE YEAR OF THE ANNALS OF SURGERY

With the issue for January, 1934, the Annals of Surgery will enter upon its fiftieth year of publication as a "Monthly Review of Surgical Science and Practice."

It will still bear upon its title page as its Editor, the same name which its first number bore, Dr. Lewis Stephen Pilcher, who will continue to actively determine its contents. Its publishers, The J. B. Lippincott Company, have made it in the past a model of typography, a proper setting for its important work. They pledge for the future to lessen no whit its high standard.

The first monthly periodical to be published in the English language devoted exclusively to Surgery, it was the outcome and the representative of the new Surgery that sprang from the union of Anesthesia with Pathology, attended by Antisepsis. During all the years since it has exhibited in a high degree the spirit of the best Surgical effort. It enters upon its fiftieth year with enthusiastic plans for the continued realization of that spirit and a full appreciation of and deep gratitude for the approval which it has received from the surgeons of the World, and with ambitions to make its future excel its past.

J. B. Lippincott Company.

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